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Micronutrient Deficiency Disorders

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Throughout the developing world high incidence of disease is associated with inadequate intake or absorption of micronutrients. Relatively minute quantities of each of the micronutrients are required for normal health status and well-being. If these nutrients are deficient, there are serious consequences for health, mental and physical. Because of known prevalence rates of deficiencies and ways of addressing them, we will focus in this chapter on three micronutrients: iron, iodine, and vitamin A. Deficiencies in other micronutrients are biologically significant—such as the effect of zinc on growth in certain disease states (Nishi and others 1980; Daeschner and others 1981), and the effect of vitamin B-6 on cellular immunity (Talbot, Miller and Kerkulirt 1987; Tomkins and Watson 1989)—but we know little about the extent of these deficiencies or how to attack them. Our purpose in this chapter, therefore, is to focus on iron, iodine, and vitamin A deficiencies and to assess global significance, public health importance, methods of prevention, and priorities within an economic framework in which costs, cost-effectiveness, and cost-benefit analyses are used. Much of what we learn about iron, iodine, and vitamin A will be useful in addressing other essential deficiencies in the future.

We will begin by discussing the global and public health significance of deficiencies in these micronutrients, including the magnitude and distribution of the deficiencies, their causes, and the implications for human health and development. We will then describe what is currently known about short- and long-term strategies and the proportion of potential beneficiaries actually covered by these programs. In the final section we will discuss issues for governments and other agencies in setting priorities for operations, institutions, allocation of resources, and research.

Public Health Significance

Micronutrient deficiencies are manifested by an array of disorders that have serious social, private, and economic costs to society.

Iron Deficiency

Iron is a mineral present in the body as a constituent of hemoglobin and in some enzyme and electron carriers. Because it cannot be made in the body, iron, like all essential nutrients, must be obtained from food.

MAGNITUDE AND DISTRIBUTION. It is generally thought that iron deficiency anemia is the most common nutritional deficiency in many developing countries, second only to protein-energy malnutrition (PEM) (Florentino and Guirriec 1984). Age and physiological status determine the degree of vulnerability of the individual: rapidly growing infants, children, and pregnant and lactating women are at high risk for deficiency. The Subcommittee on Nutrition of the Administrative Committee on Coordination (ACC/SCN) of the United Nations estimates that 1.3 billion people suffer from iron deficiency anemia (United Nations 1990). In table 19-1 this figure is disaggregated by region. Table 19A-1 contains available information on the prevalence of iron deficiency anemia by country, and table 19A-2 includes definitions for iron deficiency by age and sex.

CAUSES. Severe iron deficiency results in anemia (low hemoglobin level), which impairs the transport of oxygen and basic cell functions. People with mild iron deficiency may not have low hemoglobin (Hb) levels but yet have reduced body iron stores (ferritin) (Shils and Young 1988). "Iron deficiency" refers to any depletion of ferritin. An individual may be iron deficient without manifesting iron deficiency anemia, but all those with iron deficiency anemia are iron deficient. Anemia can also result from other nutrient deficiencies (for example, folate or vitamin B-12) or genetic abnormalities (for example, thalassemia). Folate deficiency is very commonly associated with iron deficiency anemia, so therapy usually includes a combined iron-folate tablet.

In populations in which the prevalence of iron deficiency anemia is high, the deficiency usually is the result of the

Table 19-1. Estimated Prevalence of Anemia by Geographic Region, Age, and Sex, 1980

Country	Children 0–4 years		Children 5–12 years		Men 15–59 years		Pregnant women 15–49 years		All women 15–49 years	
	Percent	Number (millions)	Percent	Number (millions)	Percent	Number (millions)	Percent	Number (millions)	Percent	Number (millions)
Africa	56	48.0	49	47.3	20	23.4	63	11.3	44	46.8
Latin America	26	13.7	26	18.1	13	12.8	30	3.0	17	14.7
East Asia[a]	20	3.2	22	5.6	11	6.1	20	0.5	18	8.4
South Asia	56	118.7	50	139.2	32	123.6	65	27.1	58	191.0
Developing regions[a]	51	183.2	46	208.3	26	162.2	59	41.9	47	255.7

Note: Anemia is defined as a hemoglobin concentration below WHO reference values for age, sex, and pregnancy status.
a. Excluding China.
Source: DeMaeyer and Adiels-Tegman 1985.

interaction between dietary factors, chronic iron loss due to parasitic infections (for example, hookworm or schistosomiasis [Stephenson 1987]), or elevated needs (for example, during pregnancy and periods of rapid growth).

It is currently believed that diet is the most important factor determining iron status. Dietary factors include insufficient iron in the diet and poor bioavailability of dietary iron. There are two types of iron in foods: heme iron (present in animal flesh), of which about 20 to 30 percent is absorbed, and nonheme iron (present in plant sources), of which less than 5 percent is absorbed. Iron in breast milk is highly bioavailable, and up to 50 percent is absorbed (INACG 1979). People in developing countries derive most of their iron from nonheme sources, whereas those in industrial countries consume greater amounts of heme iron. The key dietary difference in iron status is its bioavailability rather than the absolute amount of iron in foods (INACG 1989). Absorption of nonheme iron can be improved up to 18 percent (DeMaeyer 1989) by the addition to the diet of ascorbic acid or foods containing ascorbic acid (Hunt and others 1990) or other acids, the addition to the diet of foods containing heme iron , or the removal from the diet of substances that inhibit iron absorption (DeMaeyer 1989). Some types of food processing, such as the fermentation of soy products, also seem to improve iron bioavailability (Macfarlane and others 1990). The recommended intake of iron for people by age and sex is given in table 19A-3. The upper value is needed by people who consume mainly nonheme iron.

Although parasitic infections also contribute to iron deficiency, the treatment of such infectious parasites alone is not the most cost-effective means of addressing iron deficiency anemia, because unless the parasites are removed, reinfection will take place. Hygiene education, footwear, and improved water supply and sanitation are needed (DeMaeyer 1989).

IMPLICATIONS FOR HUMAN HEALTH AND DEVELOPMENT. It had been assumed that iron deficiency in pregnant women did not put the fetus at risk because the fetus would have priority access to maternal iron stores (Bothwell and Charlton 1981). In a study in Benin, however, Hercberg and others (1987) found that when multiple indicators of iron status were used to assess maternal anemia there was a positive correlation between maternal and infant iron deficiency. Increased prenatal and perinatal risk (low birth weight, prematurity, and mortality) has been associated with low levels of hemoglobin and hematocrit in the mother (Murphy and others 1986; Lieberman and others 1987, 1988; Brabin 1988).

In infants (6–24 months) and preadolescent children (9–11 years), iron deficiency anemia is associated with mild growth retardation (Lozoff 1982; Aukett and others 1986; Chwang, Soemantri and Pollitt 1988). Treatment of iron-deficient anemic infants (age 17–19 months [Aukett and others 1986]) and preadolescent children (age 8.2–13.5 years [Chwang, Soemantri, and Pollitt 1988]) has resulted in increased growth during the period of intervention. The causes of this growth retardation may be related to the general role of iron as an essential metabolic cofactor, its relation to immunocompetence (Higashi and others 1967; Klebanoff 1970; Chandra 1973; Mata 1977; Chandra and Puri, 1985; Dallman 1987), or its role in appetite, which decreases during iron deficiency (Basta and others 1979).

Iron deficiency anemia poses a developmental risk for cognitive dysfunction (for example, attention and concentration) in preschool and school-age children, and that risk factor is sufficiently severe to jeopardize educational attainment. The strongest and most consistent evidence of the effects of iron deficiency on cognition is found in clinical trials of preschool and school children who have been assessed for specific mental processes (for example, attention and concept formation) and school achievement. One example is a study by Soewondo, Husaini, and Pollitt (1989), which showed that anemic children three to six years old learned faster and formed appropriate concepts more efficiently after they were supplemented with iron than their placebo-fed anemic controls. In another set of studies, researchers in India (Seshardri and Gopaldas 1989) showed that when anemic children age five to fifteen were supplemented with iron, they performed better on tests for intelligence quotient (IQ), memory, visual perceptual organization, and clerical tasks improved than did anemic children who received a placebo.

Similar effects have been observed in the mental development scale scores of iron-deficient anemic infants (Lozoff and Brittenham 1985; Pollitt 1987). Iron-deficient anemic infants

perform more poorly than iron-replete infants on the Bayley Scale of Mental and Motor Development (Lozoff 1989; Walter 1989), but the behavioral response to iron therapy is not consistent across different studies (Lozoff and Brittenham 1985; Lozoff and others 1982; Oski and others 1983; Aukett and others 1986; Lozoff 1989). No changes in mental test performance have been found among infants and children whose iron stores and circulating iron are depleted but whose Hb levels have remained constant. Conceivably, these changes are too subtle to be detected in small samples.

Higher morbidity has been noted in anemic pregnant women (Fleming 1989). Reasons for this may be that iron deficiency influences the risk of infection in distinct ways. It is associated with abnormalities in cell-mediated and nonspecific immunity (Higashi and others 1967; Klebanoff 1970; Chandra, 1973; Prasad 1979; Chandra and Puri 1985; Dallman 1987). The production of T cells is specifically compromised (Srikantia and others 1976; Bagchi, Mohanram, and Reddy 1980), and the capacity of neutrophils to kill bacteria is significantly diminished during iron deficiency (Walter and others 1986). Excess free iron in the serum has been associated with predisposition to infection, but this state results largely from injected iron rather than ingested iron (DeMaeyer 1989).

Because iron deficiency anemia compromises immunocompetence, it is likely to increase mortality among high-risk groups. This increase may not be attributable to immunodeficiency alone but also to circulatory failure (INACG 1989). The exact role that iron deficiency plays in mortality needs further definition.

There is a good deal of evidence that relates maternal mortality to severe anemia. In Maharashtra, India, 90 percent of all maternal deaths occurred in women with Hb levels of less than 7 grams per hundred milliliters of blood (Masani 1969, cited in Fleming 1989). In Nigeria, Harrison (1975) found that 4 percent of mothers with severe anemia (Hb levels of less than 5 grams per hundred milliliters of blood) died in childbirth. Some evidence suggests that 20 percent of all maternal deaths in West Africa and India (when blood transfusion was not available) were directly attributable to anemia and that additional mortality resulting from hemorrhage was indirectly caused by maternal anemia (Fleming 1989).

Iron deficiency impairs work performance through its effects on Hb and, possibly, myoglobin, which is involved in the transport of oxygen in muscle. There is a high negative correlation between Hb levels (grams of Hb per 100 milliliters of blood) and the percentage of increase in heart rate of a person on a treadmill (Gardner and others 1977). A strong positive relationship exists between Hb levels and potential maximum workload as measured in the Harvard Step Test. Performance improves after iron supplementation increases Hb to the expected normal level (Scrimshaw 1984).

Iron deficiency anemia adversely affected the work productivity of Indonesian rubber tappers until they received iron supplementation (Basta and others 1979). In a recent study in Indonesia, Suhardjo (1986) found that, following iron treatment, iron-deficient anemic women increased their productivity in picking tea leaves. In Kenya, Stephenson and others (1985) found an association between the intensity of the infection with *Schistosoma haematobium*, a direct cause of iron deficiency anemia, and physical fitness among school-age children.

Iodine Deficiency

Iodine, a mineral, is a component of two thyroid gland hormones which are necessary for normal metabolism.

MAGNITUDE AND DISTRIBUTION. The number of people estimated to be at risk of disorders from severe iodine deficiency is 680 million in Asia, 227 million in Africa, and 60 million in Latin America (see table 19-2). Prevalence is high in mountainous and flood-prone areas where iodine-deficient soils prevail. Although no age group or sex is immune to iodine deficiency, the fetus, women, and children seem to be most vulnerable to serious and irreversible consequences of deficiency (Hetzel 1988). A detailed disaggregation of the prevalence of iodine deficiency by country is given in table 19A-4.

CAUSES. The term "iodine deficiency disorders" (IDDs) covers the breadth of sequelae and is not limited to severe deficiency (Hetzel 1983). Iodine deficiency disorders result from inadequate intake of iodine either because the soils and water are iodine deficient or because certain naturally existing "goitrogens" in foods interfere with the individual's use of iodine. Iodine is essential for the formation of thyroid hormones [thyroxine (T_4) and 3,5,3′-triiodothyronine (T_3)], which are necessary for normal growth and development and for proper metabolic function. Recommended daily intake of iodine is about 150–300 micrograms (see table 19A-5). When the thyroid gland does not obtain enough iodine to make these hormones, it increases in size to compensate for the deficiency. The enlarged thyroid gland is called goiter.

IMPLICATIONS FOR HUMAN HEALTH AND DEVELOPMENT. Iodine deficiency affects reproduction. Some types of anovulation can be reversed by desiccated thyroid, which confirms the relation-

Table 19-2. Estimated Prevalence of Iodine Deficiency Disorders and Population at Risk, by Region
(millions)

Region	*At risk*	*Goiter*	*Overt cretinism*
Southeast Asia	280	100	4.0
Rest of Asia	400	30	0.9
Africa	227	39	0.5
Latin America	60	30	0.3
Eastern Mediterranean	33	12	—
Total	1,000	211	5.7

— Negligible.
Source: WHO 1990.

ship, known even in ancient times, between the thyroid gland and fertility (McMichael, Potter, and Hetzel 1980). In animals there is evidence of a significant increase in spontaneous abortions and stillbirths and a marked reduction in brain growth in the fetus when the mother is iodine deficient (Hetzel and Potter 1983). Conclusive evidence of these effects on human reproduction is weak; however, reduction in stillbirths and perinatal mortality and increased birth weight in Zaire (McMichael, Potter, and Hetzel 1980; Thilly 1981, as cited in Hetzel 1987), Papua New Guinea, Ecuador, and Peru (Clugston and others 1987) have been observed after implementation of iodine deficiency control programs. In Zaire the infant mortality rate for mothers given iodine supplementation during pregnancy was significantly less than for those who were not given iodine (Thilly and others 1980).

Severe iodine deficiency in utero can result in postnatal dwarfism, and people with goiter can be retarded in their physical development (Hetzel 1988). In addition to hindering growth, iodine deficiency also increases morbidity rates in children, especially from respiratory infection (Tomkins and Watson 1989). Phagocyte dysfunction (Chandra and Au 1981) and delayed immune response (Marani,Venturi, and Nasala 1985) have been reported.

It is known that the thyroid of the fetus does not become activated until the tenth week (Delong 1987); before that, development depends on the thyroid hormone of the mother (Hetzel and Potter 1983). Deleterious effects of maternal iodine deficiency on the fetus during this time include reductions in brain DNA and RNA (Hetzel and Potter 1983). Extreme deficiency results in severe mental retardation, known as cretinism, which can take several forms. The seriousness of cretinism is obvious, but from an economic development perspective the greatest concern in endemic areas is the possibility that even noncretinous children may be mentally and neurologically handicapped. This milder impairment might remain unnoticed within the community, but it can limit the social and economic growth of these communities (Stanbury 1987).

In endemic areas the performance of noncretinous children in cognitive and motor tests correlates positively with the mother's thyroid levels during the pregnancy of the respective offspring. For example, in the Western highlands of Papua New Guinea, serum thyroxine (T_4) during pregnancy was related to the offspring's performance at twelve years of age on tests of visual perceptual organization and visual motor coordination (Pharoah and others 1984).

Given the serious effects of iodine deficiency on the proper functioning of the brain, it is not surprising to learn that even mild deficiency may have irreversible effects. A study in the highlands of Bolivia of goitrous children age five and a half to twelve years failed to show a clear effect of iodized oil supplementation given twenty-two months earlier on IQ, visual motor coordination, and school performance (Bautista and others 1982). In contrast, the authors of a study in the Guizhou province of China (Yan-You and Shu-Hua 1985) of the effects of iodized salt on the hearing of otherwise normal seven- to-eleven-year-old children one, two, and three years after prophylaxis found significant positive effects of the intervention. The difference in these outcomes is probably related to the nature of the particular function of the central nervous system that was assessed and to the mechanism and timing of the deficiency. Some hearing loss associated with acquired hypothyroidism is correctable by increased iodine intake. Still, mild mental retardation resulting from impaired structural development of the brain during fetal life is probably irreversible. Thus, addressing iodine deficiency in reproductive-age women is of highest priority.

Because of the relation between IDD and intellectual capacity, productivity is adversely affected by iodine deficiency. After a salt iodization program in one Chinese village, the average income increased from 43 yuan per person in 1981 to 223 in 1982 to 414 in 1984, which was higher than the average for the district (Levin 1987). In addition, because mental and physical fitness improved, cereals were exported for the first time and men were fit enough to join the army after salt iodization. In Ecuador, Greene (1977) found that people with moderate iodine deficiency were consistently paid less for agricultural work than normal individuals. In summary, iodine deficiency has been associated with impaired reproduction, severe and mild mental retardation, growth inhibition, and reduced productivity.

Vitamin A Deficiency

Like all essential vitamins, vitamin A is an organic substance which the body cannot produce. Vitamin A is essential for normal vision, growth, and immune function and to maintain epithelial cells.

MAGNITUDE AND DISTRIBUTION. An estimated 42 million children under the age of six have mild or moderate xerophthalmia (West and Sommer 1987). About 250,000 to 500,000 children go blind annually and approximately 50 to 80 percent of those that go blind die within one year (Sommer 1982; IVACG 1989). In tables 19-3 and 19A-6 the prevalence of vitamin A deficiency is given by region and country. The age groups at highest risk for vitamin A deficiency are young children beyond weaning age (six months to six years), although older children and pregnant and lactating women are also affected. Prevalence peaks among two- to-four-year-old children (Eastman 1987). In some parts of the world it appears that boys are at higher risk than girls (Sommer 1982; Tielsch and Sommer 1984; DeMaeyer 1986), which may be a reflection of different cultural practices in rearing children or physiological differences (Sommer 1982). Prevalence is also greatest in low-income groups and during those seasons when food sources of the vitamin are scarce (Mamdani and Ross 1988). In table 19A-2, definitions for vitamin A deficiency are listed. Note that the population percentages in this table refer to those with severe vitamin A deficiency. There are currently no values for mild vitamin A deficiency.

CAUSES. Xerophthalmia and its cure through the diet were recognized in ancient times. It is only in the early part of this

Table 19-3. Estimated Prevalence of Vitamin A Deficiency, 1984–85

Region	Countries with deficiency[a]	Children aged 1–4 years (millions)	Children with mild to moderate deficiency[b] (millions)
Africa	16	53	7.9
Americas	5	38	5.6
Southeast Asia[c]	5	51	7.7
India	1	111	16.7
Mediterranean	3	7	1.0
Western Pacific	4	20	3.0
Total	34	280	41.9

a. Countries in WHO category A (significant problem with control programs in place 1984–85): Bangladesh, El Salvador, Haiti, India, Indonesia, Nepal, Philippines, Sri Lanka. Category B (significant problem but no control program 1984–85): Benin, Brazil (northeast states), Burkina Faso, Ethiopia, Malawi, Mali, Mauritania, Mexico, Oman, Sudan, Tanzania, Viet Nam, Zambia. Category C (probable problem but no assessment or program 1985–85): Afghanistan, Angola, Bolivia, Burma, Chad (north), Ghana (north), Kampuchea, Kenya, Lao P.D.R., Mozambique, Niger, Nigeria (north), Uganda.

b. Assumes 15 percent prevalence.

c. Excluding India.

Source: West and Sommer 1987.

century, when vitamin A was discovered, that the deficiency was first described in connection with physical growth and, later, with vision. In the more recent past greater attention has been placed on the association between the deficiency and infant and child morbidity and mortality. Traditionally, vitamin A deficiency has been defined as a severe reduction in vitamin A reserves along with clinical signs of the deficiency. Milder depletion may also be defined as deficiency, even though it does not result in changes in the eyes, because it may still have an important relationship to morbidity and mortality (West and Sommer 1987). Biochemically, even mild signs of deficiency in children are detected by a decrease in vitamin A reserves, with liver and serum levels less than 20 micrograms per deciliter (see table 19A-2).

Dietary sources of vitamin A include preformed vitamin A (retinol) from animal sources and beta-carotene and other carotenoids found in plant sources, which can be converted to vitamin A in the body. Retinol is the most active form of vitamin A, followed by beta-carotene and then the other carotenoids (see table 19A-5 on how these three compare). Vitamin A deficiency is caused by dietary inadequacy (see table 19A-5 for recommended intakes), by increased physiological requirements, and by cultural factors which determine individual availability and consumption. In countries in which the staple food is rice, a cereal without vitamin A, low-income groups are at high risk of vitamin A deficiency. Even in countries in which sources of the carotenoids exist, there may be high incidence of deficiency. In parts of Indonesia, for example, dark green, leafy vegetables (a rich source of beta-carotene) are commonly available, yet vitamin A deficiency is highly prevalent. This coincidence is partly explained by the low social value attributed to green vegetables (WHO 1982, as cited in Mamdami and Ross 1988); partly by an inadequate source of fat, which facilitates the absorption of the carotenoids (United Nations 1985); and partly by protein inadequacy, which hinders vitamin A absorption, release, and transport (Mamdami and Ross 1988). Vitamin A transport is affected only when protein deficiency is severe (Sommer 1982). Intestinal abnormalities caused by bacterial infection or parasites can also interfere with the absorption of vitamin A. Respiratory infection and other diseases can increase the requirement for the vitamin and interfere with its intake through decreased appetite (United Nations 1985). In Africa, measles is often a precipitating factor in blindness due to vitamin A deficiency (Eastman 1987).

In addition, there are social and economic factors related to the deficiency (Mamdami and Ross 1988). For example, in urban Bangladesh a clear negative association has been noted between per capita income and vitamin A deficiency (Stanton and others 1986). Within low-income groups, however, such a correlation is often less obvious. Intrafamily food distribution patterns and, as noted, the low social value attributed to foods rich in vitamin A may determine risk in vulnerable groups.

Seasonal availability of vegetables and fruits often acts synergistically with other factors to precipitate deficiency. In countries such as the Philippines and Indonesia, dark green, leafy vegetables are generally unavailable when infections peak.

IMPLICATIONS FOR HUMAN HEALTH AND DEVELOPMENT. When pregnant women are deficient in vitamin A, severe xerophthalmia may develop in utero (Sommer 1982), which increases vulnerability to infection and death. Vitamin A deficiency also decreases fertility (Eastman 1987). Congenital malformations have also been related to experimentally induced vitamin A deficiency in animals. Less is known about the effects of vitamin A deficiency on congenital malformations in human beings, although some suggestive clinical observations exist (Wallingford and Underwood 1986), such as congenital xerophthalmia, anophthalmia, microphthalmia, and other ocular defects (IVACG 1986). Vitamin A overdose during early pregnancy may cause fetal absorption (Eastman 1987). For this reason it is generally recommended to give pregnant women no more than 1,000 micrograms of vitamin A at a time or to treat existing deficiencies with dietary sources of vitamin A (IVACG 1986).

The ocular signs of vitamin A deficiency fall under two categories: (a) night blindness due to the interruption of the dark adaptation process in the visual cycle; and (b) structural changes in the surface of the eye (Bitot's spots, drying, keratomalacia, ulceration, and so on) due to loss of secretory function in the mucosal epithelium in the conjunctiva of the eye and changes in the differentiation or maturation of specific epithelial cell types (Mamdami and Ross 1988). Night blindness, conjunctival dryness, and Bitot's spots are generally reversible, but more advanced stages are not and can result in lesions which cover the entire cornea, causing partial or total blindness. Once the disease has reached this extreme level of severity, the life of the child is endangered.

Both mild and severe forms of vitamin A deficiency are associated with increased morbidity, especially from respiratory and diarrheal disease (Sommer, Katz, and Tarwotjo 1984). The effects of vitamin A deficiency on cell-mediated immunity, antibodies, and secretory antibodies have been documented using both animal models and clinical data (Nauss 1986; Olson 1986; Chandra 1988). Conclusive information on its adverse effects on phagocytosis is not available.

It is known that measles and vitamin A deficiency have a complex reciprocal interaction. Vitamin A status is a determinant of the outcome of measles, especially in Africa, and measles, in turn, is a forceful precipitating factor in blindness (Eastman 1987). For example, measles was related to bilateral corneal ulceration in 78.9 percent of these cases (Ksanga, Pepping, and Kavishe 1985, as reported by Eastman 1987). In fact, the peak for vitamin A deficiency coincides with the peak for measles in children (Eastman 1987). An association between vitamin A deficiency and measles has also been found in Asia. In studies in Bangladesh (Cohen and others1985) and Indonesia (Sommer 1982), 10 percent and 37 percent, respectively, of children with keratomalacia had had measles within the previous four weeks. The intensity of the infection, and not a specific characteristic of measles, seems to be the determining factor in the high mortality observed among vitamin A deficient children (WHO/UNICEF 1987; WHO/EPI 1988b). The release of vitamin A from storage in the liver is apparently hindered by infection (DeMaeyer 1986). The effect on the immune system explains, in part, a well-documented association between vitamin A deficiency and growth retardation in animals (McLaren 1966, cited in West and Sommer 1987; Eastman 1987). In experimental animals vitamin A causes a cessation of bone growth along with loss of appetite (Eastman 1987).

Although conclusive information on mortality risks among children with all levels of vitamin A deficiency is not yet available (Wittpenn and Sommer 1986), it has been estimated that of the children with keratomalacia who remain untreated, 60 percent will die (West and Sommer 1987). Some studies show that even children with mild vitamin A deficiency have higher mortality rates than matched controls (Sommer and others 1983; Sommer and others 1986; Rahamathullah and others 1990). The cause of this is probably related to vitamin A's role in maintaining healthy mucosal tissue throughout the body and in the immune function. To confirm these findings similar studies are under way in other countries, and results should provide conclusive evidence on whether vitamin A deficiency increases the mortality risk in children (National Academy of Sciences 1987).

There are no known effects of vitamin A deficiency on the growth and development of the brain and intelligence. Blindness or partial blindness would obviously affect learning, especially in the classroom setting. To the extent that it increases morbidity, even mild vitamin A deficiency may affect school performance and productivity. At issue here is how many preschool- or school-age children with a history of xerophthalmia are left out of formal schools because of blindness due to vitamin A deficiency (Pollitt 1990). This question is particularly troublesome in Africa, where corneal scarring associated with measles is frequent (WHO/EPI 1988b). In Malawi and Tanzania, for example, half of the children attending schools for the blind reported a history of measles preceding total blindness (WHO/EPI 1988b).

To summarize, vitamin A deficiency has been linked with interference with ocular function, impaired growth and reproduction, and increased morbidity and mortality. Deficiencies in the three micronutrients are manifested by overlapping conditions. In table 19-4 we review the deficiency conditions for all three and show the similarities in the conditions for the micronutrients.

Micronutrient Interactions

The interaction of micronutrients can be viewed in two different ways. Deficiencies of the three micronutrients under discussion interact according to their geographic setting and according to how the micronutrients are metabolized.

Iron, iodine, and vitamin A deficiencies often occur in countries in which poverty limits dietary sources and in which geography limits the composition of food that normally would contain these micronutrients. Generally, these three micronutrient deficiencies occur simultaneously in certain areas of Africa, the Andes of South America, and in many parts of Asia (see tables 19A-1, 19A-4, and 19A-6).

Metabolism of the micronutrients may also be affected by dietary components. Protein-energy malnutrition interferes with iodine metabolism (Ingenbleek and De Visscher 1979; Gaitan, Mayoral, and Gaitan 1983). Other nutrients may also increase or inhibit the absorption or use of these micronutrients. Vitamin A and zinc deficiencies might interact synergistically (Baly and others 1984). Vitamin A deficiency also affects anemia (Bloem and others 1990). For example, in Guatemala, fortification of sugar with vitamin A resulted in improvement of hemoglobin levels (Mejia and Arroyave 1982). Because vitamin A seems to affect hemoglobin levels but not body stores, it might be involved in the synthesis of hemoglobin and red blood cells (Mejia and Chew 1988). Other components in foods may inhibit the use of dietary micronutrients, such as phytates in some plants, which inhibit iron absorption.

Prevention

Adequate consumption of the micronutrients through food is the best way of preventing micronutrient deficiencies. Recommended daily intake by age and sex is presented in table 19A-3 (iron) and table 19A-5 (iodine and vitamin A).

When the intake of these foods is limited, specific interventions are needed to prevent and address micronutrient deficiencies. Most micronutrient interventions represent both preventive and curative therapies. High-dose supplements, in particular, can be used to treat severe deficiency and to prevent deficiency in vulnerable age groups.

Table 19-4. Functional Effects of Essential Micronutrient Deficiencies

Effect	Deficiency		
	Iron	Iodine	Vitamin A
Morbidity			
Immune function	Yes	Unknown	Yes
Prevalence	Yes	Yes	No
Incidence	No	Unknown	Yes
Duration	No	Unknown	Yes
Severity	No	No	Yes
Mortality			
Infant	Yes	Yes	Yes
Child	Unknown	Yes	Yes
Maternal	Yes	Unknown	No
Other (fetal, early adult)	No	Yes	No
Mental development and learning disorders			
Brain development	Unknown	Yes	No
Aptitude	Yes	Yes	No
Intelligence quotient	Yes	No	No
Exploratory behavior	Yes	No	No
Attention span	Yes	Yes	No
Memory	Unknown	Yes	No
School achievement	Yes	Yes	Unknown
Learning disability (blindness, deafness)	No	Yes	Yes
Sensory impairment	Yes	Yes	No
Productivity			
Spontaneous activity	Yes	Yes	No
Endurance	Yes	No	No
Maximum aerobic capacity	Yes	No	No
Occupational productivity	Yes	Yes	No
Disability[a]	No	Yes	Yes
Growth	Yes	Yes	Yes
Reproduction			
Fertility	No	Yes	Yes
Miscarriage and stillbirth	Yes	Yes	Yes
Intra-uterine growth retardation	Yes	Yes	No
Prematurity	Yes	No	No
Congenital deformities (birth defects)	No	Yes	Yes

a. Such as blindness, mental retardation, or lack of motor coordination.
Source: Iron—DeMaeyer 1989; Iodine—Hetzel, Dunn, and Stanburg 1987; Vitamin A—West and Sommer 1987, Eastman 1987.

There are two main types of interventions to reduce micronutrient deficits: supplementation (the administration of pills, capsules or injections containing one or more of the micronutrients) and fortification (the addition of micronutrients to foods in processing). Other interventions, such as nutrition education and agricultural programs, can be used over the long term to promote the intake of these micronutrients by vulnerable groups.

Supplementation

Delivery of micronutrients through supplementation can be done in a variety of ways. The supplements can be taken orally or by injection. Typical iron supplementation programs are shown in table 19A-7. Some supplements must be taken daily (such as oral iron), whereas others can be taken at intervals of three to five years, as is the case for iodized oil. The frequency depends mainly on the ability of the body to store the micronutrient (substantial in the case of iodine and vitamin A).

Although iron deficiency is usually treated with supplements from one of several iron compounds, ascorbic acid increases the absorption of nonheme iron from existing sources like maize, rice, wheat, or sorghum, in which the iron content is adequate but is in an unabsorbable form. Vitamin C supplementation and fortification can therefore be considered to be an iron intervention. When taken with such foods, ascorbic acid can increase the absorption of available iron by about 30 percent (Hallberg 1981; Berg and Brems 1986), or from about 5 percent to about 6.5 percent.

A significant challenge for prophylaxis or treatment through supplementation is compliance (taking the proper dosage at appropriate intervals). This is a problem particularly

in iron supplementation, which requires the daily ingestion of iron tablets, sometimes with mild side effects (headache, nausea, and so on) in the initial weeks of supplementation. Side effects were thought to be an important detractor to compliance, but in recent iron studies in Thailand and Burma, only 10 to 15 percent of women taking iron complained of side effects (Charoenlarp and others 1988), and only a small proportion of those women failed to take supplements because of side effects. In another study, Griffiths (1980) found that when women were warned of possible side effects they were more likely to continue taking their iron supplements when side effects occurred. Ensuring compliance with iron therapy has been most successful in situations in which supplements are provided and ingestion is supervised, such as in the workplace and schools. But for persons who do not participate regularly in such institutions or who live in outlying areas, compliance and tablet availability are serious obstacles to success. A recent study of iron ingested in slow-release capsules (gastric delivery system [GDS]) showed that side effects did not differ between placebo, ferrous sulfate, and GDS. In addition, compliance did not seem to differ between ferrous sulfate or GDS even though the dosage for GDS was one pill per day and that for ferrous sulfate was two (Simmons 1990). The GDS iron was better absorbed, however.

When available, long-lasting, megadose supplements are superior to low-dose supplementation with respect to both cost and compliance. This is particularly true for injections of iodized oil, which provide protection for as long as four to five years, and oral doses of iodized oil, which are good for two years (Underwood 1983; Berg and Brems 1986). Large doses of iodine can cause adverse reactions (thyrotoxicosis, with such symptoms as increased heart rate, trembling, sweating, and weight loss [Hetzel 1988]), although this is not generally a significant problem because in most cases spontaneous remission will occur (Medeiros-Neto and others 1987). The highest risk of thyrotoxicosis is in people over forty, so giving supplements only to younger adults avoids most of this toxicity (Berg and Brems 1986). High doses of vitamin A are both toxic and teratogenic. Great care must be taken not to give vitamin A capsules at higher dosages than recommended or to women who might be pregnant (generally 1,000 micrograms or less). Vitamin A can be toxic even in children, so an alternative delivery scheme based on low, frequent doses is being investigated (Underwood 1989). As with any essential drug, micronutrient supplements need to be handled properly to ensure their stability, potency, availability, and proper use.

Fortification

Where essential micronutrient deficiencies are prevalent, dietary fortification is generally considered preferable to supplementation as a long-term strategy in controlling micronutrient deficiencies. The advantages of dietary fortification are that compliance is ensured if the appropriate carrier is selected. The cost of delivery of the micronutrients through food staples is far less than through the health system and can be partially or fully borne by the consumer. A crucial step in fortification is choosing the right food to fortify. Several criteria are used in selecting a particular food vehicle for fortification (Beaton and Bengoa 1976; Baker and DeMaeyer 1979):

- It must be a food that is consumed by the vast majority of the target population and in adequate amounts.
- It must be able to be fortified on a large scale and at relatively few centers, so the fortification can be adequately supervised.
- It must be stable under the extreme conditions likely to be encountered in storage and distribution.
- It must not interfere with the use of the nutrient, and the nutrient must not interfere with the food (that is, it must not be detrimental to flavor, shelf life, color, texture, or cooking properties).

Table 19-5 shows typical fortification programs for the three micronutrients under discussion. The usual food vehicles are salt or sugar because they tend to meet the four criteria. The food chosen for fortification is site specific; thus successful fortification programs have included wheat flour, skimmed milk, monosodium glutamate (MSG), infant foods, beverages, salt, and condiments (Clydesdale and Wiemer 1985). Foods commonly eaten only by specific subpopulations may prove to be satisfactory vehicles for targeting high-risk groups, such as small children. For example, in many industrial countries weaning foods are fortified with iron.

The fortification of salt with iodine has been practiced extensively in industrial countries. Many Latin American countries have passed legislation requiring iodization of all salt. Unfortunately, maintaining supplies of iodized salt has remained a problem in these countries. Political commitment at all levels of government and the community is needed for such legislation to be effective. A successful national program in Bolivia organized small salt producers into cooperatives, which made them more able to compete with larger

Table 19-5. Typical Fortification Program

Nutrient	*Compound*	*Vehicle*	*Concentration*	*Source*
Iron	Ferric orthophosphate	Salt	3.5 g/kg	Working group on fortification of salt with iron 1982
	NaFe EDTA	Sugar	13 mg/100 g	Viteri and others 1981
Iodine	Potassium iodate	Salt	15–40 ppm	Mannar 1987
Vitamin A	Retinol palmitate	Sugar	10 mg/g	Arroyave and others 1979

Source: See last column.

producers. This action increased their productivity, which in turn increased the availability of iodized salt, improving consumption of iodized salt and reducing the prevalence of goiter (Pardo 1990).

Fortification may not always be feasible, however, because of the lack of an appropriate carrier food available to at-risk groups, weak enforcement of fortification regulations, (Berg and Brems 1986), or excessive cost of the fortified food. The most appropriate carrier may also be a food that has unrelated health effects, such as hypertension and tooth decay, which are linked with the overingestion of salt and the consumption of sugar, respectively. In these cases other strategies may be required. Fortification is the best method available for solving micronutrient deficiency problems in the long term because of low cost, good coverage, and technical feasibility. The success of fortification in the long term hinges crucially on the regularity and enforcement capacity in the governmental departments responsible for food safety and quality. Without effective public and private oversight of the fortification system, the quality of a fortification program can deteriorate rapidly. Sufficient incentives and penalties for private industry and public regulators need to be set up to ensure longevity.

Double fortification of foods may be a way to address two micronutrient deficiencies in a cost-effective way. India has experimented with double fortification, using iron and iodine. Although the technology has not been completely worked out, double fortification offers hope in dealing with multiple micronutrient deficiencies in areas where several coexist.

Other Interventions

While supplementation and fortification are two of the main interventions used to address micronutrient deficiencies, there are a number of other interventions—nutrition education, breastfeeding promotions, agriculture, food processing, public health measures—that should be used either alone or along with supplementation and fortification efforts.

NUTRITION EDUCATION. Education of the consumer about nutrition is an important component of any micronutrient intervention. It is needed along with every fortification, supplementation, and food production program to guarantee that the intended beneficiary actually consumes the nutrients. Failure to educate the public and politicians to support fortification programs over the long term has been implicated as a significant reason for the failure of programs in Central and South America (Schaefer 1974, as cited in Thilly and Hetzel 1980). In both Guatemala and India, mass media and communication campaigns were required to create demand for fortified salt (Thilly and Hetzel 1980; United Nations 1987).

More general nutrition education is also important to increase intake of the micronutrients from the existing food supplies. Lack of maternal knowledge of the need for children to consume leafy green and yellow vegetables is associated with increased risk of nutritional blindness (Stanton and others 1986).

Breastfeeding promotion is important for micronutrient nutriture (as well as other nutrition and health benefits) because, if the mother is in good health, it is a good source of iron, iodine, and vitamin A. In addition, a protein in breast milk, lactoferrin, reduces free iron in the intestinal lumen and hence protects the infant against infection while simultaneously rendering the iron more absorbable (Tomkins and Watson 1989). Breastfeeding promotion is also important because vitamin A deficiency has been associated with the early cessation of breastfeeding (Stanton and others 1986). In Bangladesh the risk of a child's developing one or more signs of vitamin A deficiency is six times higher for a child younger than two years of age who is not breastfed (Mamdami and Ross 1988), and length of lactation was found to influence the risk of vitamin A deficiency in children in Ethiopia (de Sole, Belay, and Zegeye 1987, as cited in Mamdami and Ross 1988) and Indonesia (Sommer 1982).

Promoting the increased production and use of nutrient-rich local foods through agricultural programs and policies (improved marketing, greater dietary diversity, increased rural incomes) may prove invaluable to any program aimed at reducing the incidence of these deficiencies. Not surprisingly, in urban Bangladesh, risk of vitamin A deficiency in children was associated with poor intake of foods rich in vitamin A (Stanton and others 1986). In Bangladesh, families without gardens are more likely to have children with xerophthalmia than are those with gardens (Cohen and others 1985).

Research and development of new technologies to increase the micronutrient content of raw and processed foods and new high-nutrient varieties are also needed. For example, in processed foods the absorption of iron could be greatly increased by the addition of ascorbic acid or by the decrease of substances that compete with iron for absorption. Public health measures to alleviate environmental factors which exacerbate dietary deficiencies (Stanton and others 1986) may be recommended also.

Program Coverage

In tables 19A-1, 19A-4, and 19A-6 we give approximations for program coverage for iron, iodine, and vitamin A, respectively. As can be seen in table 19A-4, there is much activity in planning and implementing national programs to combat iodine deficiency disorders. Control programs usually involve either fortification or supplementation. Legislation to make iodization of food mandatory is more infrequent except in some Latin American countries in which legislation was passed several decades ago. It should be noted, however, that even with the passage of legislation, goiter has persisted in these countries, which indicates the difficulty in controlling and regulating industry to comply with the law. Knowledge of coverage for these iodization programs is scant, and even in a country such as Bhutan, where 100 percent of the salt is iodized, there are questions of whether or not this salt is reaching remote endemic areas and of whether the stability of iodine in the salt can be maintained at effective levels.

Vitamin A program activity has increased dramatically over the last several years, but gaps still exist in the knowledge of actual coverage in countries. Only a few countries have undertaken vitamin A fortification programs, making supplementation the most frequent option at present. Pilot studies in the Philippines for fortifying MSG with vitamin A have proved promising (Muhilal and others 1988; Muhilal, Muherdiyantiningsih, and Karyadi 1988). As for program coverage for iodine, program coverage for vitamin A is still underreported. Until such information is available, it will be difficult to gauge progress in combating the deficiency.

Control programs for iron deficiency are not well documented, even though iron supplementation presumably is part of standard practice in prenatal care. Considering the magnitude of the deficiency problem and the effects of iron deficiency, the lack of attention given to addressing the problem is surprising. Countries with active programs, such as India, have found difficulties in population compliance with a consequence of low coverage. More than 80 percent of those dropping out of iron supplementation programs cited discontinued supplies of tablets as the reason for noncompliance (United Nations 1990). Much more needs to be done to document exact prevalence and geographic location of iron deficiency so that control programs can be effectively targeted to those most at risk. Better program design is also needed to meet present problems.

Case Management

Prevention is the best type of case management, but where severe deficiencies of iron, iodine, and vitamin A exist, case management is best handled by trained health personnel and may require hospitalization. Immediate attention should be given to correcting deficiencies upon diagnosis. The exception to this is cretinism, which is irreversible. Immediate attention should be given to the mothers of cretinous children to improve the outcomes of future pregnancies. With mild and moderate deficiency, case management can be supervised by community health workers. In table 19A-2 we show how severe deficiencies in iron, iodine, and vitamin A can be detected, and in table 19A-7 we give typical supplementation programs for all these micronutrients.

For anemic patients with circulatory failure or respiratory distress, blood transfusion is required. Because blood loss of a severely deficient person can precipitate shock or heart failure, it is of vital importance to increase the hemoglobin level of anemic pregnant women before and during labor. If a child experiences severe vomiting or diarrhea after taking oral vitamin A capsules, water miscible retinol palmitate injections can be used. Oil-based injections should not be used because they are metabolized too slowly to be effective in acute, severe deficiency (West and Sommer 1987).

Assessment of the Effect of Interventions

In order to determine the best solution for addressing micronutrient deficiencies, the prevalence, socioeconomic and geographic distribution, and causality need to be assessed in each country. Priorities for resource allocation must be based on the nature of the problem (prevalence, severity, geographic distribution, causality), the cost-effectiveness of alternative solutions, the institutional capacity to carry out the interventions, and the cultural acceptability of solutions.

Cost-Effectiveness Analysis

Different delivery systems are associated with different costs and effectiveness. In this context, the term "costs" refers to the value of all resources required to deliver the micronutrients to the target population. The term "effectiveness" refers to program and biological effectiveness. Program effectiveness is the efficacy of the delivery system in providing adequate dose and coverage to those with deficits. Biological effectiveness is the efficacy of the dose to eliminate the deficiency. Cost-effectiveness is the cost per unit of change in the outcome of interest (West and Sommer 1984). The choice of a delivery system should depend heavily upon its relative cost-effectiveness, the most desirable strategies being those with the highest effectiveness relative to cost. An additional economic criterion is that of the cost-benefit relation for each intervention. Micronutrient interventions have both costs—the value of resources required for delivery of micronutrients to the appropriate populations—and benefits—the improved functioning of those populations through the elimination of micronutrient deficiencies. For example, decreases in vitamin A deficiency will reduce blindness, allowing affected populations to care more fully for themselves, to reduce needless expenditures on health care, to benefit from education, and to be more productive in the workplace. Reduction of iron deficiencies improves educational outcomes and work output in both the household and the workplace. Reduction of iodine deficiencies decreases the likelihood of cretinism and other disorders that burden society.

One criterion for determining where to make investments in a resource-scarce situation is to allocate resources to those endeavors in which the ratio of benefits of the intervention to costs exceeds alternatives. Costs can provide a measure only of the cost of delivery and not of the program and biological effectiveness. Any measure of cost-effectiveness must take into account both the cost and the effectiveness of the intervention. The methodologies involved in determining costs and benefits are included in appendix 19B. In appendix 19C we describe the criteria of effectiveness. In appendix 19D general methodologies for cost-benefit analysis are presented.

Any comparative analysis of cost-effectiveness should take account of costs properly accounted for to meet micronutrient needs among at-risk persons for a given period of time. In table 19-6 the costs per person are given for different interventions in 1987 dollars (column 3). Column 4 presents the data from column 3 corrected for the duration of the dose. We stress in appendix B the need to use an ingredients or resource recovery method to estimate costs. Moreover, we indicate some of the reasons that the costs of a given intervention in a particular context cannot necessarily be generalized to other contexts.

With respect to micronutrient requirements, there is no assurance that different interventions have the same success for the reasons stated in appendix B. The prevalence and severity of micronutrient deficiencies differ from site to site and affect the success (and costs) of an intervention. Different interventions cover requirements for different lengths of time. A year of fortification, for example, meets one year's requirements; oral iodine covers needs for two years; injected iodine covers needs for three to five years, depending upon the dosage; and vitamin A capsules are associated with four to six months of protection, although lower-dose vitamin A supplements are available for monthly dosing. Clearly, the cost per year must be adjusted for the duration of protection provided by the intervention. Finally, the cost per year must be adjusted for the benefit leakage. In a large-scale fortification effort, not all of the recipients will be at risk, so the cost per person at risk will be higher than the cost per recipient. Targeting more finely may not be cost-effective even if leakages are high. If only one-third of the population is at risk of micronutrient deficiency, the cost per each at-risk person will be three times as high as the average cost per person.

One of these adjustments is made in the last numerical column in table 19-6, where the estimated cost per person in the previous column is calculated on the basis of one year of protection. The enormous differences in estimated cost between fortification with iodine and iodized oil injections is narrowed considerably when the five-year period of protection for oil is taken into account. Even so, the differences in cost for oil injections are substantial among studies, which may be the result of real differences, of some of the site-specific differences, or of poor data. With respect to vitamin A capsules, the two studies show considerable agreement on costs. Because two capsules must be taken per year, the cost per year of protection is twice that for the administration of a single capsule—the cost basis in each of the studies. The difference in the costs between the two iron fortification programs using sugar as the vehicle is due to the addition of ascorbic acid to the second program. Ascorbic acid costs fifteen to twenty times as much as ferrous sulphate. The high estimated cost for delivery of ferrous sulphate tablets is because of the relatively high personnel requirement for providing daily tablets and the active supervision and motivation needed to obtain compliance. If such tablets could simply be delivered every six months to households, the cost would fall much closer to the cost of distributing vitamin A capsules.

Over time the cost-effectiveness may change. Tilden and Grosse (1988) found that dietary modification programs were more cost-effective over twenty years than either supplementation or fortification programs and that supplementation was more cost-effective over time than fortification. In their analysis they showed that over a twenty-year time period dietary modification is more effective than supplementation, which is more effective than fortification in preventing blindness and death from vitamin A deficiency. It should be kept in mind that fortified foods and water are consumed by large numbers of people who are not at risk. Thus, the cost per at-risk person for supplementation and fortification is much closer than the differences shown in table 19-6, and the cost-effectiveness of the two strategies may not be very different once these adjustments are taken into account. This is especially true for vitamin A deficiency; the numbers of those at risk are small (children under five) in relation to the entire population, and thus targeted supplementation would be more cost-effective.

Table 19-6. Cost of Micronutrient Interventions

Nutrient form	*Country and year*	*Cost per person (U.S. dollars)*	*Estimated cost per person (1987 U.S. dollars)*	*Estimated cost per person per year of protection (U.S. dollars)*	*Source*
Iodine					
Oil injection	Peru 1978	1.30	2.30	0.46	Hetzel and others 1980
Oil injection	Zaire 1977	0.35	0.67	0.14	Hetzel and others 1980
Oil injection	Indonesia 1986[a]	1.00	1.05	0.21	Irie and others 1986
Water fortification	Italy 1986	0.04	0.04	0.04	Squatrito and others 1986
Salt	India 1987	0.02–0.04	0.02–0.04	0.04	Mannar 1987
Vitamin A					
Sugar fortification	Guatemala 1976	0.07	0.14	0.14	Arroyave and others 1979
Capsule	Haiti 1978	0.13–0.19	0.23–0.34	0.46–0.68	Austin and others 1981
Capsule	Indonesia and Philippines 1975	0.10	0.21	0.42	West and Sommer 1984
Iron					
Salt fortification	India 1980	0.07	0.10	0.10	Cook and Reusser 1983
Sugar fortification	Guatemala 1980	0.07	0.10	0.10	Viteri and others 1981
Sugar fortification	Indonesia 1980	0.60	0.84	0.84	Levin 1985[b]
Tablets	Kenya and Mexico 1980	1.89–3.17	2.65–4.44	2.65–4.44	Levin 1985

a. Per injection.
b. From data provided in Derman and others 1977.
Source: See last column.

An additional way of measuring cost-effectiveness across a variety of health and nutrition interventions is to use a common outcome measure and estimate the costs per unit of that outcome. Throughout this collection, the disability-adjusted life-year gained is one measure of universal applicability. It is also possible to use cost per life saved or cost per unit of economic productivity gained.

The cost-effectiveness of achieving these outcomes with micronutrient interventions is shown in the tables in appendix 19E and summarized in table 19-7. Using the current prevalence of deficiencies commonly observed in developing countries and certain assumptions about demographics, death and disability, coverage and effectiveness (75 percent), the discount rate (3 percent), and life expectancy (seventy years), we calculated the discounted cost per disability-adjusted life-year gained based on available costs of micronutrient control programs.[1] For calculations of productivity, the annual wage rate was assumed to be $500, unemployment was assumed to be 25 percent, and disabilities were assumed to be the same as the health disabilities in table 19E-1. No adjustment was made for increased cost of feeding a more productive worker or for the employment replacement effect of increased productivity.

Cost-Benefit Analysis

Cost-benefit analysis for all micronutrients, although performed on a limited number of countries, suggests that both supplementation and fortification are good investments.

IRON. Levin (1985) estimated the benefits and costs of both medicinal supplementation and dietary fortification with iron-deficient populations on the basis of data from Indonesia, Kenya, and Mexico. Benefits accrued primarily from higher work output associated with normalization of hemoglobin levels in anemic populations. A remarkable degree of consistency was found in eight studies of work output related to hemoglobin levels. The elasticity of work output with respect to rises in Hb was between one and two: that is, an increase in Hb of 10 percent was associated with a rise in work output of 10 to 20 percent.

The pecuniary value of additional work output was estimated by first ascertaining the probable rise in Hb associated with particular interventions. This rise in Hb was converted into an increase in individual work output by applying the elasticities from the studies mentioned above. Because at least some of the rise in individual work output will simply replace the work output of others in a labor surplus economy, the social benefits will be less than the sum of the individual increases in productivity. That is, some workers will no longer be needed and will be unemployed as the output of other workers increases. Therefore, only half of the increase in work output was assumed to be a net increase in social productivity.

The net increase in social productivity was valued according to the wages that would be required to produce that additional output. The total value of productivity was adjusted to a per capita level by dividing by the entire population, including the portion of the population that was not economically active. Finally, the results were adjusted for the estimated effects of improved iron status on outputs other than work productivity. These include lower morbidity and mortality, greater physical stature, higher productivity outside of the workplace, improved quality of leisure time, greater learning and faster school advancement, and increased feelings of well-being. Especially important is the additional work output in the household and in peasant agriculture, which is not accounted for by the value of additional output in labor markets. This adjustment raised the value of total benefits by 50 percent above those of just the market-based work benefits.

Costs were estimated for fortification strategies in which both salt and sugar were used as examples of dietary vehicles. Supplementation was based on the assumption that iron supplements were one of four different dietary or health interventions delivered by a health system. Both the average cost per intervention was estimated as well as the marginal cost of the iron supplements alone. The cost of fortification was based on data from field trials. The cost of supplementation was based on the use of village health auxiliaries, various modes of transportation, facilities, and the cost of the supplements. An additional cost in both types of interventions resulted from the higher energy needs of workers who produce a larger work

Table 19-7. Return on Nutrition Investments

Intervention	Cost per life saved (U.S. dollars)	Discounted value of productivity gained per program dollar	Cost per disability-adjusted life-year gained (U.S. dollars)
Iron deficiency			
Supplementation of pregnant women only	800	24.70	12.80
Fortification	2,000	84.10	4.40
Iodine deficiency			
Supplementation of reproductive-age women only	1,250	13.80	18.90
Supplementation of all people under sixty years	4,650	6.00	37.00
Fortification	1,000	28.00	7.50
Vitamin A deficiency			
Supplementation of children under five years only	50	146.00	1.40
Fortification	154	47.50	4.20

Source: Based on table 19E-1.

output. This cost was estimated on the basis of the additional calorie input required for the additional work output, calculated from the cost of the additional rice or cornmeal required to produce those calories.

The benefits and costs were compared under a wide range of scenarios, including Hb increases, elasticities of work output with respect to Hb changes, costs of the interventions, and so on. Benefits in relation to costs were found to be highest in Mexico and lowest in Indonesia, with Kenya occupying the middle position. In all cases and under all reasonable conditions the benefits of both fortification and supplementation exceeded the costs by a wide margin. Under an intermediate set of conditions—neither the most optimistic nor the most conservative—the benefit-cost ratio for fortification varied from seven to one for Indonesia to forty-two and seventy to one for Kenya and Mexico, respectively. For supplementation, including the costs of both the ferrous sulphate and the prorated costs of the delivery system, the intermediate range of benefit-cost ratios was four, twenty-three, and thirty-eight to one for Indonesia, Kenya, and Mexico, respectively. Even under the most pessimistic assumptions, the benefits of the interventions exceeded their costs, often substantially.

IODINE. In table 19-8 we provide an overall map of the various documented effects on both human and animal populations of reductions in iodine deficiency disorders as well as the various benefit categories. In theory, it is only necessary to translate the effects into benefits and to place monetary values on them to compare them with the costs of an intervention. Unfortunately, the lack of field trials that incorporate data collection in the various benefit domains limits the application of cost-benefit analysis to this area. Nevertheless, two benefit-cost studies for iodine interventions provide illustrations of what appear to be substantial benefits in relation to costs. Correa (1980) did a benefit-cost analysis of reducing mild iodine deficiency in children. Benefits were assumed to be based on the higher earnings associated with reducing the mental effects of iodine deficiency, whereas costs were derived for the interventions necessary to reduce iodine deficiency. He first used data from a variety of sources to assess the relation between iodine deficiency and the IQ of children. He then used an independent source of data on the relation between IQ and earnings for a sample of adult men in Chile to project the earnings effects for the children with higher IQs due to iodine sufficiency. According to his calculations, the benefits, seen as improvements in lifetime earnings, of reducing mild iodine deficiency among children exceeded considerably the costs of the interventions; however, we should be cognizant that the estimates are based on diverse data sets and heroic assumptions that connect them.

More recently, the authors of two studies of screening and treatment of congenital hypothyroidism in the United States (Barden and Kessel 1985; ICCIDD/WHO 1989) showed benefits equal to three times the costs when benefits were the savings in institutional care, foster care, special education, productivity losses, and other requirements of caring for a retarded child. Both studies are limited with regard to a comprehensive understanding of benefits and costs associated with IDD control in those areas of the world with substantial at-risk populations, but they do suggest a high payoff from iodine interventions.

VITAMIN A. A relatively comprehensive cost-benefit study of a vitamin A intervention for reducing xerophthalmia was undertaken by Popkin and others (1980). The study was based on Philippine data in which the social benefits of reducing xerophthalmia were viewed as increased income and the reduction in costs of outpatient care because fewer children would die, go blind, or become sick due to the disease. The assumption was that xerophthalmia affects both the future productivity and the development of children ages one through fifteen. Higher mortality and total blindness from xerophthalmia lower the productive time, and increased morbidity, mortality, and partial blindness reduce future productivity. Reductions in prevalence also reduce treatment costs. Three types of interventions were analyzed for their costs and effectiveness in reducing xerophthalmia: mass dose capsules of vitamin A every six months; fortification of MSG with vitamin A; and a program of health and nutrition education, disease prevention through sanitation and immunization, and limited curative work. The benefits were found to be substantially greater than costs for the mass dose capsule and MSG fortification interventions, but costs exceeded benefits for public health interventions. For the mass dose capsule, benefits were from 2.4 to 3.4 times the costs. For fortification, the benefits were 6 to 21 times the costs. As mentioned previously, Tilden and Grosse (1988) found that over time, long-term programs, such as dietary modification, will be more cost-effective than shorter-term interventions, such as supplementation and fortification.

Strengthening Institutional Capacity

Many different institutions can be used to control micronutrient deficiencies. The most obvious is the infrastructure of the

Table 19-8. Effects of Iodine Interventions

Population	*Physiologic effects*	*Benefits to society*
Humans	*Reductions in:* Mental deficiency Deaf-mutism Spastic diplegia Squint Dwarfism Motor deficiency Goiter	Higher work output Reduced costs of medical and custodial care Reduced educational costs because less absenteeism and grade repetition and higher achievement
Livestock	*Increases in:* Live births Weight Strength Health[a] Wool coat in sheep	Higher output of meat and other animal products Higher work output of animals

a. Less deformity.
Source: Hetzel and Maberley 1986; Levin 1987.

primary health care system, which is particularly important for supplementation programs. Expanded programs in immunization have been suggested as a vehicle for delivering supplements of vitamin A (WHO/EPI 1988a). Because vitamin A deficiency peaks in children two to four years of age, use of the existing Expanded Programme on Immunization (EPI) infrastructure may in fact make a lot of sense. Conversely, it may be preferable to put the supplementation in the hands of a village health worker. In that case the low-dose pump bottle may be the preferred delivery vehicle for vitamin A because the risk of overdosing is much lower. In some countries, health systems have low coverage, making it necessary to attack the problem differently. Nongovernmental organizations may also integrate micronutrient interventions into their programs, making an important contribution to the reduction of deficiencies in the groups in which they work. An alternative may be the school system, which can deliver micronutrients to the general community as well as children. Delivery to children is particularly relevant in view of the evidence that deficiencies in iodine, iron, and vitamin A affect the ability to learn. School feeding programs may increase attendance in schools and thus may increase the coverage of micronutrient interventions run through schools.

Markets can be used to distribute micronutrients either through fortification of food staples or increased agricultural production of micronutrient-rich food sources. The supply of micronutrients can be increased and, through consumer and nutrition education, demand and use can be expanded. Governments need to establish food regulatory institutions to ensure that fortification laws are being enforced.

Priorities for Resource Allocation

The most efficient way to allocate resources may be to give priority to vulnerable populations based on age, sex, and geographic region. Iron interventions should be targeted first to pregnant women and preschool children; vitamin A interventions should be targeted primarily to preschool children, but also to school-age children and pregnant and lactating women; and iodine interventions should be targeted to women of reproductive age and children.

The selection of a particular micronutrient intervention should be based on severity of deficiency, budgetary resources, and institutional capacity. Short-term solutions to frank deficiencies usually involve supplementation, which is easily targeted and quickly administered, but more comprehensive programs are needed (fortification, nutrition education, and food production). Food fortification, if it is feasible, will normally be the most cost-effective, long-term option in populations in which a high proportion is at risk because it entails only the marginal cost of adding the nutrient to a food staple, modifying the packaging, if necessary, to protect potency, and enforcing regulations. These marginal costs of fortification could be borne by producer, consumer, government, or some combination of these.

In some countries, fortification is not feasible because a suitable carrier cannot be identified. In others, regions that are populated by at-risk populations are so remote that transportation routes are not adequate to provide regular and continuing supplies of the fortified food. In both cases, mass dose supplements, fortification of the water supply, and the growth of nutrient-rich foods provide a basis for localized micronutrient programs.

The cost of supplementation is generally higher than that of fortification because of delivery, monitoring, and counseling. Supplements can be delivered by mobile health teams that visit villages periodically or by the primary health system and essential drug program.

Supplementation costs can be reduced by targeting programs only to those at risk and incorporating supplementation into existing programs, including EPI (WHO/EPI 1987). The compliance problem with iron supplements could be overcome by assigning a village health worker to provide health education to the villages with special emphasis on increasing the intake of the micronutrients.

Priorities for Operations Research

Although technology and operational experience are sufficient to implement micronutrient deficiency control programs now, more applied research is needed to improve the assessment and treatment of micronutrient deficiencies. Research is also needed on the oral supplements themselves. A slow-release iron supplement is needed which would obviate the need to take tablets every day. Development of a slow-release vitamin A supplement would minimize toxicity problems. Similarly, the technical system for fortifying salt with both iron and iodine is needed.

Another set of research issues relates to measuring the prevalence of micronutrient deficiencies. Surveys of micronutrient deficiencies are often outdated with respect to both the data themselves and the methodology used. The improved assessment of iodine status (thyroid stimulating hormone) from a drop of blood on filter paper can replace highly subjective goiter surveys and unreliable urinary excretion assessments. A new measure of iron status (transferrin receptors) exceeds the specificity of all other measures and can be done using a finger prick sample.

Further epidemiological research is needed on the incidence of multiple micronutrient deficiencies. Greater cost-effectiveness can be attained if ways are found to prevent more than one deficiency at a time. The potential of using a filter paper blood sample to analyze all three of the deficiencies under discussion would accelerate this research.

Much more information is needed about cost-effectiveness and institutional support. Although the data on iron are fairly strong, cost-effectiveness and cost-benefit data on vitamin A and iodine programs are weak. Effective enforcement of fortification programs needs further research, including the nature of legislation, the structure of regulatory bodies, and the mechanisms for inducing compliance. Finally, better data are needed on the functional effects of micronutrient deficiencies, especially those relating moderate deficiency to human resource development.

Summary and Conclusions

Iron, iodine, and vitamin A deficiencies are highly prevalent in developing countries. The existing data are sufficiently robust to conclude that these deficiencies result not only in increased mortality but also in increased morbidity, learning disabilities, reproductive wastage, and reduced work output. Yet cost-effective solutions exist and have been successful under a wide spectrum of conditions; supplementation and fortification are feasible but their appropriateness depends on local characteristics.

It is important to engage in long-term planning to control micronutrient deficiencies because the need will not diminish in the near future. Strategic planning requires an active assessment of institutional capacity and of the cost-efficiency of alternative methods. Sustainability is a central concern in designing any micronutrient deficiency control program. If supplementation or nutrition education are used, they need to be integrated into the health care system. Specific and targeted campaigns may work, but they are too costly in relation to integrating strategies into a more comprehensive health care approach. Micronutrients should not displace but rather complement protein-energy malnutrition as a key problem for health workers. Special attention should be given to organizational strengthening, including strong regulatory structures, if we are to win the battle against micronutrient deficiency disorders.

Appendix 19A. Prevalence, Programs, Recommended Intakes, and Indicators of Deficiencies

The tables in this appendix present detailed information on the iodine, iron, and vitamin A deficiencies discussed earlier in the chapter. Country-by-country statistics, which have been provided wherever possible, include data on current interventions.

Table 19A-1. Prevalence of Iron Deficiency and Program Coverage by Country

		Fortification		
Country	*Prevalence (percent)*	*Program status*	*Legislation*	*Program status*[a]
Africa				
Benin	39.0[b]	None	None	None
Burundi	7.2[b]	None	None	None
Chad	25.0[b]	None	None	None
Ethiopia	6.0[b]	None	None	None
Kenya	6.0[b,c]	None	None	None
Mali	4.6	None	None	None
Sierra Leone	50.0[b]	None	None	None
Tanzania	25.0[b,c]	None	None	None
Zambia	49.0[b]	None	None	None
Zimbabwe	45.0[b]	None	None	None
Asia				
Bangladesh	66.0[c]	None	None	None
Burma	82.0[b]	None	None	None
China	86.0[b,c]	None	None	None
India	69.0[b]	None	None	Under way[d]
Indochina	37.0[b]	None	None	None
Malaysia	83.0[b]	None	None	None
Nepal	24.1[b]	None	None	None
Philippines	37.5[b]	None	None	Under way[e]
Sri Lanka	3.8[b]	None	None	None
Thailand	11.0[b]	None	None	None
Middle East				
Algeria	41.9[b]	None	None	None
Egypt	22.4[b]	None	None	None
Lebanon	32.0[b]	None	None	None
Morocco	11.2[b]	None	None	None
Pakistan	20.0[b]	None	None	None
Syrian Arab Republic	47.0[b]	None	None	None
Tunisia	29.9	None	None	None
Latin America				
Argentina	16.0[b]	None	None	None
Bolivia	18.5[b]	None	None	None
Chile	20.0[b]	Under way	None	Under way
Costa Rica	7.0	None	None	None
Cuba	30.0[b]	None	None	None

(Table continues on the following page.)

Table 19A-1 (continued)

Country	Prevalence (percent)	Fortification: Program status	Fortification: Legislation	Program status[a]
Latin America (continued)				
Ecuador	46.0[b]	None	None	None
El Salvador	8.6[b]	None	None	None
Guatemala	National[f]	Under way	None	None
Guyana	40.0[b]	None	None	None
Jamaica	76.0[b]	None	None	None
Paraguay	76.0[b]	None	None	None

Note: The quality of data (sample size, age, region) necessitates caution when comparing countries.
a. Many countries distrubute iron tablets as part of MCH programs, but there are little data on coverage, compliance, effect, and so forth.
b. Regional or sporadic prevalence, or known regionally in some age groups and sexes.
c. Probable.
d. Twelve percent coverage.
e. Forty-five percent coverage.
f. Iron deficiency anemia prevalent nationally. Exact numbers not available.
Source: Chafkin 1984; Levin 1986; Hercberg and others 1987; Yepez and others 1987; Stekel 1987; Assami and others 1988; Prual and others 1988; Valyasevi 1988; Pollitt 1989; FAO undated; Florentino, undated; Seshadri and Gopaldas, undated.

Table 19A-2. Definition of Deficiencies

Group		Value
Iron deficiency	*Hemoglobin (g / dL)*[a]	+
Children six months to six years		11.0
Children six to fourteen years		12.0
Adult males		13.0
Adult females, nonpregnant		12.0
Pregnant females		11.0
All ages	*Serum ferritin (mcg / L)*	< 10–12
Iodine deficiency	*Goiter prevalence (percent)*	*Median urinary iodine (mcg/g creatinine)*
Mild		
Population	5–20	n.a.
Individual	n.a.	> 50
Moderate		
Population	21–29	n.a.
Individual	n.a.	25–50
Severe		
Population	> 29	n.a.
Individual	n.a.	< 25
Vitamin A deficiency		
Population: children younger than six years (percent)		
Night blindness (xN)[c]	1.0	
Bitot's spots (xIB)[c]	0.5	
Corneal scars, ulceration (x3A)[c] 0.01		
Xerophthalmia-related corneal scars	0.05	
Serum retinol < 10 mcg/dL	5.0	
Individual	*Serum retinol (mcg/dL)*	*Liver retinol (mcg/g)*
Mild deficiency		
Children	20	< 20
Adults	30	< 20
Severe deficiency		
Children	< 10	< 10
Adults	< 10	< 10

n.a. Not Applicable.
a. Hemoglobin values below which anemia is likely to be present in individuals living at sea level.
b. Prevalence of endemic cretinism is 1–10 percent.
c. Clinical abbreviation for stage of Xerothphalmia.
Source: West and Sommer 1987; DeMaeyer and Adiels-Tegman 1985; Hetzel 1988; DeMaeyer 1989.

Table 19A-3. Recommended Daily Intake of Iron, Based on Bioavailability in Diet

Age group	Absorbed iron requirement (mg/day)	Iron intake (mg/day) by quality of the diet[a]		
		Low bioavailability	Medium bioavailability	High bioavailability
4–12 months	0.96	32	16	9
13–24 months	0.61	20	10	5
2–5 years	0.70	23	12	6
6–11 years	1.17	39	19	11
Girls 12–16 years	2.02	67	34	18
Boys 12–16 years	1.82	61	30	16
Adult Males	1.14	38	19	10
Adult Females				
Pregnant	3.6	120	60	33
Lactating	1.31	44	22	12
Menstruating	2.38	79	40	22
Postmenopausal	0.96	32	16	9

a. As defined by Monsen and others 1978, a diet with low bioavailability contains no meat, fish, or poultry; none of the iron is heme iron, and 3 percent of total iron is absorbed. A diet with medium bioavailability contains 1 ounce of fish per day; four percent of the iron is heme iron, and 6 percent of total iron is absorbed. A diet with high bioavailability contains 3 ounces of beef per day; 21 percent of the iron is heme iron, and 11 percent of total iron is absorbed.

Source: DeMaeyer 1989.

Table 19A-4. Prevalence of Iodine Deficiency and Program Coverage, by Country

Country	Prevalence		Fortification			Supplementation program status
	Percent	Area[a]	Program status	Legislation (years)	Coverage (percent)	
Africa						
Angola	—	Regional	None	None	—	None
Benin	—	Regional	None	None	—	None
Botswana	63	Regional	None	None	—	Planned
Burkina Faso	7.7	National	None	None	—	Planned
Burundi	56	Regional[b]	Under way	None	—	Planned
Cameroon	59	Regional	None	None	—	Planned
Central African Republic	25	Regional	None	None	—	None
Chad	11	Regional	None	None	—	None
Comoros	40	Regional	None	None	—	None
Congo	—	Regional	Under way	1988	—	None
Côte d'Ivoire	18	Regional	None	None	—	Planned
Ethiopia	34	Regional	Under way	None	—	Under way
Gabon	1	National	None	None	—	None
The Gambia	—	Regional	None	None	—	None
Ghana	13	Regional	None	None	—	Planned
Guinea	15.4	National	None	None	—	None
Guinea Bissau	—	Regional	None	None	—	None
Kenya	15–72	National	Under way	1970	50	Planned
Lesotho	14.3	National	Planned	None	—	Planned
Liberia	—	Regional	None	None	—	Planned
Madagascar	18	Regional	None	None	—	Planned
Malawi	30–70	Regional	Planned	None	—	Under way
Mali	20	Regional	None	None	—	Planned
Namibia	—	Regional	None	None	—	None
Niger	13	National	None	None	—	Planned
Nigeria	40	Regional	None	None	—	Planned
Rwanda	19	Regional	Under way	None	—	Planned
Senegal	33	Regional	Under way	None	—	None
Sierra Leone	—	Regional	None	None	—	Planned
Somalia	—	Regional	None	None	—	None
Sudan	20	Regional	None	None	—	Planned
Swaziland	26	National	None	None	—	None
Tanzania	40	Regional	Under way	None	—	Under way
Togo	—	Regional	None	None	—	Planned
Uganda	—	Regional	None	None	—	None
Zaire	—	Regional	None	None	—	Under way
Zambia	27–81	Regional	Under way	1979	—	Planned
Zimbabwe	20	Regional	None	None	—	Planned

(Table continues on the following page.)

Table 19A-4 (continued)

Country	Prevalence		Fortification			Supplementation program status
	Percent	*Area*[a]	*Program status*	*Legislation (years)*	*Coverage (percent)*	
Asia						
Bangladesh	10.5	National	Under way	1989	55	Under way
Bhutan	64.5	National	Under way	None	100	Under way
Burma	14.3	National	None	None	—	Under way
Cambodia	30	National	None	None	—	Planned
China	—	Regional	Under way	None	87	None
India	7.3	National	Under way	1962	12	Planned
Indonesia	20	National	Under way	1976	51	Under way
Korea, Republic of	—	—	None	None	—	Planned
Lao PDR	—	—	None	None	—	Planned
Malaysia	—	Regional	Under way	None	0	None
Nepal	46.1	National	Under way	None	72	Under way
Papua New Guinea	40	Regional	Under way	1972	—	Planned
Philippines	14.9	National	Under way	None	—	Planned
Sri Lanka	19.3	National	Planned	None	0	Planned
Thailand	14.7	National	Under way	None	2	None
Viet Nam	34	National	Under way	None	5	None
Middle East						
Afghanistan	—	National	None	None	—	None
Algeria	—	Regional	Under way	None		Planned
Egypt	70	Regional	None	None	—	None
Iran	60	Regional	None	None	—	Planned
Iraq	80	Regional	None	None	—	Planned
Lebanon	50	Regional	None	None	—	Planned
Libya	20	Regional	None	None	—	Planned
Morocco	—	Regional	None	None	—	None
Pakistan	—	Regional	Under way	None	11–17	None
Tunisia	—	Regional	None	None	—	None
Latin America						
Argentina	15.6	National	Under way	1967	99	Under way
Bolivia	61	National	Under way	1967	20–80	Under way
Brazil	14.7	Regional	Under way	1977	—	None
Chile	18.8	Regional	Under way	1968	85	Under way
Colombia	1.8	Regional	Under way	1947	—	None
Costa Rica	3.5	National	Under way	1970	—	None
Cuba	30.3	Regional	None	None	—	Under way
Dominican Republic	80	Regional	None	None	—	None
Ecuador	36.5	Regional	Under way	1968	75–80	Under way
El Salvador	48	National	Under way	1961	17	Under way
Guatemala	10.6	National	Under way	1954	36	Under way
Honduras	17	National	Under way	1961	—	Under way
Mexico	—	Regional	Under way	1962	—	Under way
Nicaragua	20	National	Under way	1969	—	Under way
Panama	6	National	Under way	1966	47	Under way
Paraguay	18.1	National	Under way	1966	—	Under way
Peru	50–80	Regional	Planned	None	60	Under way
Uruguay	—	Regional	Under way	1961	—	Under way
Venezuela	21.3	National	Under way	1968	—	Under way

— Data not available.

Note: The quality of data (sample size, ages, region) necessitates caution when comparing countries.

a. Regional or sporadic goiter prevalence, or known regionally in some age groups; or national goiter prevalence or prevalent nationally in some age groups.

b. Probable.

Source: FAO no date; Hetzel 1987; United Nations 1987; PAHO/WHO/UNICEF 1988; Dunn (ed.) 1989a and 1989b; ICCIDD 1989; ICCIDD/WHO 1989; "Iodine Deficiency" 1989; Pollitt 1989; "Status of IDD" 1989; WHO 1989.

Table 19A-5. Recommended Daily Intake of Iodine and Vitamin A
(mcg/day)

Age group	*Intake*
Iodine	
All People	150–300
Vitamin A	
Infants younger than 1 year	300
1–3 years	250
4–6 years	300
7–9 years	400
10–12 years	575
13–15 years	725
16–19 years	750
Adult men and women[a]	750
Lactating women (first six months)	1,200

Note: Micrograms or retinol equivalents (RE) are currently used to measure retinol and the carotenoids. Previously, international units were used. To convert: 1 RE = 1 mcg retinol = 6 mcg all-trans beta-carotene = 12 mcg other provitamin A carotenoids = 10 IU provitamin A carotene = 3.33 IU of retinol.

a. Including pregnant women and lactating women after the first six months.

Source: FAO 1965; Hetzel 1988.

Table 19A-6. Prevalence of Vitamin A Deficiency and Program Coverage by Country

	Prevalence		*Fortification*		*Supplementation*
Country	*Percent*	*Area*[a]	*Program status*	*Legislation*	*program status*
Africa					
Angola	—	Probable	None	None	None
Benin	3.5	Regional	None	None	None
Botswana	—	Regional	None	None	None
Burkina Faso	—	National	None	None	None
Burundi	—	Probable	None	None	Under way
Chad	—	National	None	None	None
Côte d'Ivoire	—	Regional	None	None	Under way
Ethiopia	—	Regional	None	None	None
Ghana	—	National	None	None	Under way
Kenya	—	Probable	None	None	Under way
Madagascar	—	Regional	None	None	None
Malawi	3.9	Regional	None	None	Under way
Mali	—	National	None	None	Under way
Mauritania	2.3	Regional	None	None	Under way
Mozambique	—	National	None	None	Under way
Niger	—	National	None	None	Under way
Nigeria	—	Regional	None	None	Under way
Rwanda	—	Probable	None	None	None
Senegal	—	Regional	None	None	None
Somalia	11.0	Regional	None	None	Under way
Sudan	1.6	Regional	None	None	Under way
Tanzania	1.6	Regional	None	None	Under way
Uganda	—	Regional	None	None	Under way
Zaire	—	Regional	None	None	Under way
Zambia	—	Regional	None	None	Under way
Zimbabwe	—	Regional	None	None	None
Asia					
Bangladesh	4.9	National	None	None	Under way
Burma	—	Regional	None	None	Under way
Cambodia	—	Probable	None	None	None
China	—	National	None	None	None

(Table continues on the following page.)

Table 19A-6 (continued)

Country	Prevalence		Fortification		Supplementation program status
	Percent	Area[a]	Program status	Legislation	
Asia (continued)					
India	12–20	National	None	None	None
Indonesia	20.0	National	Under way	None	Under way
Lao PDR	—	Probable	None	None	None
Malaysia	—	Regional	None	None	None
Micronesia	10.0	n.a.	None	None	None
Nepal	1.0	National	None	None	Under way
Philippines	—	National	Planned	None	Under way
Sri Lanka	—	Regional	None	None	Under way
Thailand	—	Regional	None	None	None
Viet Nam	—	National	None	None	Under way
Middle East					
Afghanistan	—	Probable	None	None	None
Algeria	—	Regional	None	None	None
Egypt	—	Regional	None	None	None
Iran	—	Regional	None	None	None
Iraq	—	Regional	None	None	None
Jordan	—	Regional	Under way	1977	None
Morocco	—	Regional	None	None	None
Pakistan	—	Probable	None	None	Under way
Syrian Arab Republic	—	Regional	None	None	None
Yemen, Republic of	—	Regional	None	None	None
Yemen, Arab	0.57	Regional	None	None	None
Latin America					
Bolivia	—	Regional	None	None	None
Brazil	—	Regional	None	None	Under way
Costa Rica	—	Regional	Under way	Under way	None
Ecuador	—	Regional	None	None	None
El Salvador	—	Regional	None	None	Under way
Guatemala	—	Regional	Under way	Under way	Under way
Haiti	0.81	Regional	None	None	Under way
Honduras	—	Probable	Under way	Under way	None
Jamaica	—	Regional	None	None	None
Mexico	—	Regional	None	None	None
Nicaragua	—	Regional	Under way	None	None
Panama	—	Regional	Under way	Under way	None
Peru	—	Regional	None	None	None

— Data not available.

Note: The quality of data (sample size, ages, region) necessitates caution when comparing countries.

a. Either regional or sporadic prevalence or known regionally, or prevalent nationally, with clinical signs in children younger than six years.

Source: Arroyave 1982; Cohen and others 1985; FAO no date; IVACG 1989; Mathur and Kushwaha 1987; Pollitt 1989; Solon and others 1983; Underwood 1983; UNICEF 1988; United Nations 1985; WHO 1988a and 1988b.

Table 19A-7. Typical Supplementation Programs

Deficiency	Target group	Compound	Dose	Frequency
Iron				
Presumptive treatment	Pregnant women	Ferrous sulfate	200 mg	Twice daily
where prevalence of	Pregnant women	Folic acid	500 mcg	Daily
iron deficiency anemia	Children six months to five years[a]	Ferrous sulfate	10 mg/kg	Daily[b]
is moderate or high	School-age children	Ferrous sulfate	200 mg	Daily
Presumptive treatment	Pregnant women	Ferrous sulfate	200 mg	Daily
where prevalence of	Pregnant women	Folic acid	250 mcg	Daily
iron deficiency anemia	Children six months to five years	Ferrous sulfate	3 mg/kg	Daily[b]
is mild	School-age children	Ferrous sulfate	100 mg	Daily
Treatment of severe iron deficiency anemia	Pregnant women	Ferrous sulfate	200 mg	Thrice daily for four weeks
(hemoglobin less than 7 g/dL)	Pregnant women	Folic acid	250 mcg	Thrice daily for four weeks

Deficiency	*Target group*	*Compound*	*Dose*	*Frequency*
	Children six months to five years	Ferrous sulfate	10 mg/kg	Daily
	Adults	Ferrous sulfate	200 mg	Twice daily
Treatment of moderate	Pregnant women	Ferrous sulfate	200 mg	Twice daily
iron deficiency anemia	Pregnant women	Folic acid	250 mcg	Daily
(hemoglobin between	Children six months to five years	Ferrous sulfate	3 mg/kg	Daily
7 and 10 g/dL)	Adolescents	Ferrous sulfate	200 mg	Twice daily
	Adults	Ferrous sulfate	200 mg	Twice daily
Iodine	All	Iodinated oil-oral	2 ml	Two years
	All	Iodinated oil[c]	2 ml	Three to five years
	All	Iodinated oil-intratranscular	1 ml	Three years
Vitamin A	Children without xerophthalmia	Oil solution	20,000 mcg	Three to six months after age one year
	Children with xerophthalmia[d]	Oil solution	20,000 mcg	Diagnosis
			20,000 mcg	Next day
			20,000 mcg	Two to four weeks later, at clinical deterioration, or at discharge
	Pregnant women without dietary sources	Oil solution	1,000 mcg	Daily
	Lactating women	Oil solution	20,000 mcg	At parturition
			1,000 mcg	Daily

Note: All treatment is oral, unless otherwise specified.

a. Children younger than four months should be given only breast milk, which provides adequate iron. After four months, iron-containing weaning foods should be given. Low-birthweight infants require iron from two months age.

b. Short-course therapy.

c. Injected. A caveat for use of any injections is increased risk of AIDS transmission where needles are commonly reused and probability of sterilization is low.

d. Children younger than twelve months should receive half doses.

Source: IVACG 1986; Dunn 1987; West and Sommer 1987; DeMaeyer 1989; United Nations 1990.

Appendix 19B. Costs of Supplementation and Fortification

Before we review the costs of supplementation and fortification, it is important to review briefly the appropriate method for measuring costs. Although the notion of costs is often used quite casually in the health sector literature, it has a very specific meaning in the economics literature (Mishan 1976; Levin 1983; Mills 1985). It refers to the social value of all the resources, or ingredients, that are required to provide an intervention—even resources provided in kind. The proper method for ascertaining costs is first to specify the particular resources that are needed for a nutritional intervention, such as the facilities, personnel, materials, and micronutrients that are required. Second, the value or cost of each of the ingredients is derived from both market data, if these are available, and other determinants of economic value, such as shadow price (Mills 1985). These costs for all the ingredients are summed to determine the total cost of an intervention. The total cost can be divided by the overall population or another base to obtain the cost per participant of the intervention.

The determination of the cost of an intervention is independent of the issue of how it is financed. The cost is a measure of the value of resources that are used for the intervention, no matter who is paying for them. Although an analysis of who pays or should pay and how it should be financed is important, it is the subject of a separate analysis. It is important to get an accurate determination of the cost before addressing its financing.

Cost Estimation

Using the ingredients method of determining the cost of the delivery of medicinal supplements, such as iron, one must first select a delivery model. The usual model is that of a community- or village-based health care system in which there is a heavy use of local resources, such as health auxiliaries or community health workers. There is a considerable literature on village or community health workers (Djukanovic and Mach 1975; Hetzel 1978; WHO 1979; Bender and Yoder 1983). Such workers are people who have completed all or most of primary school and are literate in basic reading, writing, and computational skills. They typically come from the local community, so they relate well to the populations they serve. They can deliver nutritional supplements and provide information and advice on their use. They are able to offer inoculations and other specific health services for which they are qualified through short training programs, and they periodically have contact with more highly trained staff. Other ingredients for the supplementation model include facilities, equipment,

transportation, and the micronutrient supplements themselves (Hetzel and others 1980; West and Sommer 1984; Levin 1985, 1986).

The costs of the fortification intervention are based on those of the micronutrient compounds as well as personnel, equipment, and special packaging required for the preparation and delivery of the fortified product (Arrojave and others 1979). The value of the food vehicle that is being fortified is not a cost of fortification but only the additional cost associated with the fortification process. To protect the micronutrient content associated with fortification, some products, such as fortified salt, must be packaged in more expensive bags than their unfortified counterparts.

Comparing Costs

The literature on micronutrient interventions contains cost estimates for both supplementation and fortification. Usually, these costs are expressed as the cost per person covered by the intervention. Thus, in theory, it would appear that one could readily compare the costs of injected supplements for iodine or vitamin A with the costs of orally administered supplements. Or one could compare the costs of fortification of different food vehicles with iodine, such as salt or water, or with iron, such as salt, sugar, wheat flour, or milk products. Then one could select those interventions that had the lowest cost per person for delivery.

For example, one of the most obvious features of table 19-6 is the large differences in the cost per person, even when standardized to 1987 dollars. Even for a single intervention, such as injections of iodinated oil, the cost per person varied from $0.67 to $2.30. The costs per person among different interventions for the same micronutrient differ even more.

Idiosyncratic Differences in Costs

In fact, comparisons of costs among studies cannot be used as a basis for determining the most efficient form of supplementation or fortification. The reason is that the costs in any particular study will—to some degree—be idiosyncratic because of the methodology used and the time and setting in which it was carried out (Mills 1985). It is important to summarize the sources of these idiosyncrasies to show why cost results from one study cannot necessarily be compared with another.

- *Methodological differences*. Different studies use different methodologies, from casual guesses at costs to rigorous cost accounting methods. Even among the latter there are differences in assumptions and methods based on different judgments. Unless a uniform method is used among studies, comparison is inappropriate.
- *Exchange rates*. The usual practice is to convert costs in local currencies to some standard monetary unit, such as U.S. dollars, that can be compared among studies. But exchange rates are distorted by speculation, government intervention, dominance of particular commodity flows, and other factors that do not reflect the true value of resources in that standard monetary unit. The result is that some of the cost differences among interventions drawn from the experience of different nations may depend on fluctuating exchange rates that are not in competitive market equilibrium rather than on the "true" world value of those resources.
- *Time*. The usual cost comparisons among studies do not take account of the fact that often the studies were carried out at different times. Because prices change over time and follow different patterns among countries and because there are often no appropriate price deflators for standardizing them over time, the costs derived at different time periods are not strictly comparable even after adjusting for price-level changes.
- *Context*. A cost comparison between two different countries and situations will reflect the unique characteristics of those contexts. For example, it costs far less for health teams to go from village to village in a relatively flat region with good transportation than it does for them to travel in mountainous or jungle regions. Differences in population density affect costs profoundly because of the difficulties of reaching sparse and remote populations as compared with more compact ones. Tropical climates may pose additional costs for packaging and refrigeration of micronutrients than more temperate climates. The result is that some of the measured cost differences among interventions may derive from the differences in circumstances rather than intrinsic differences in costs.
- *Local price differences*. Some differences in costs among interventions in different countries and regions are due to differences in prices for the same commodities and labor. For example, the price of iodinated oil was found to be about 50 percent higher in Bolivia in 1985 than in France in 1983, a difference that cannot be explained by inflation (Dunn 1987). Among three countries in 1979, the cost of persons trained as vaccinators varied from $2.24 a day in Indonesia to $5.90 a day in Thailand, a considerable difference (Creese and others 1982). This is another reason that cost experiences in one country for a particular intervention might not reflect the costs of that intervention in another country.
- *Population base*. Some of the interventions are targeted to only the populations at high risk, such as is the case of iodinated oil in villages with a high incidence of goiter and low iodine in the soil. Others, such as the iodination of salt, are based on distributing a fortified product to the entire population, those in need of the intervention and those not in need. If only one-third of the population is in need of a particular intervention, the cost per person at risk is three times as high as the cost per capita in the population as a whole. Thus, the cost per person of interventions that reach only those who are at risk should not be compared with the much lower costs of those that reach the entire population, including those who are not at risk.

Appendix 19C. Criteria of Effectiveness

Some interventions will have a high success rate in obtaining repletion, such as injected or oral iodinated oil or oral capsules of vitamin A. Once ingested or injected, these interventions are almost invariably associated with iodine or vitamin A repletion. In contrast, medicinal supplementation with iron or dietary fortification does not always ensure repletion. Because the capacity of the body to store iron is limited, iron supplementation requires that the participant take iron daily. When administered in schools or workplaces, this compliance can be readily maintained. When it is necessary to depend on households continually to take iron supplements, it is not realistic to expect a high level of compliance. Thus, the cost of delivering the iron to households is not equivalent to the cost of obtaining iron repletion. Indeed, obtaining compliance may require continuing reinforcement through monitoring and persuasion by village health teams and other educational efforts.

The same is true with fortification. Not only is it necessary for all persons at risk to consume adequate amounts of the fortified food, but the food must have sufficient amounts of the micronutrient at the time of consumption. There may be a compliance problem when unfortified, local products compete with the nationally or regionally distributed fortified ones. In Ecuador it was necessary to mount a social marketing campaign to increase use of a fortified product such as iodinated salt because alternative salt sources were available at the local level (Manoff 1987). In tropical areas the hygroscopic nature of salt that is used for iodine fortification means that unless contained in watertight packaging until consumption, at least some of the iodine will be lost. Iodinated salt in jute bags showed a loss of three-quarters of its iodine in nine months (Mannar 1987). The type of packaging, the time it takes to get to consumers, and the use of open or closed containers by shops and consumers will determine potency. In very humid climates with highly undependable transportation and long periods before sale or consumption in open containers, the salt may lose virtually all its iodine.

Appendix 19D. Cost-Benefit Analysis

Cost-benefit analysis represents a technique for ascertaining whether micronutrient and other social interventions are worthwhile. Such interventions use scarce societal resources, which could be used to provide other types of social benefits. At a minimum, an intervention should not be undertaken unless its benefits exceed its costs. But because many investments have benefits that exceed costs, it is also important to consider whether the relation of benefits to costs exceed those—or are at least in the range of—alternative investments.

Although it would be desirable to have a standard cost-benefit methodology with precise rules for calculation for every situation, this is not the present case. The cost methodology is straightforward and is identical to the ingredients, or resource recovery, method that was outlined in appendix 19B for cost and cost-effectiveness studies. But although the conceptual methods for identifying and measuring benefits are well established (Creese and Henderson 1980; Mills 1985), the application of these methods depends crucially on a variety of judgments on both the measurement of benefits and their values. Some of the best work on cost-benefit analysis in the health sector is found in the area of immunization (Creese and Henderson 1980; Creese 1983), and many of the methods used there can be applied to micronutrients.

The basic method of estimating benefits is to identify the positive effects of micronutrient interventions on such areas as morbidity, work output, and educational benefits for children. The benefits of reduced morbidity are generally considered to be the savings in health care and the value of lost productivity; the benefits of work output can be measured with respect to additional days of productive work (in the labor market or household) and the additional productivity per day; and educational benefits include the value of additional student achievement and the reduction in the cost of special educational services or grade repetition. Some of these benefits also have implications for costs. For example, if iron-replete workers are able to put out more work effort to increase productivity, they will also need additional food to compensate for the higher expenditure of energy (Levin 1985, 1986).

As summarized in the earlier sections of this chapter, each of the micronutrient interventions has an effect on health, productivity, and other aspects of behavior. In theory, it is only necessary to translate the effects into benefits and to place monetary values on them to compare them with the costs of an intervention. Unfortunately, the lack of field trials that incorporate data collection in the various benefit domains limits the application of cost-benefit analysis to this area. Nevertheless, there exist studies for each of the three micronutrients that are both informative and suggest high returns. These are discussed in the main text of the chapter.

Appendix 19E. Costs and Benefits

The tables in this appendix show the costs and benefits of various interventions.

Table 19E-1. Assumptions in Calculating Costs per Disability-Adjusted Life-Year, Death Averted, and Income Enhancement

Parameter	*Value*
Program effectiveness (percent)	75[a]
Unemployment (percent)	25[b]
Life expectancy (years)	70
Discount rate (percent)	3
Annual wage rate (U.S. dollars)	500
Population (number)	100,000
Age distribution (number)	
0–1 year	3,900
1–2 years	3,250
2–3 years	2,340
3–4 years	1,950
4–5 years	1,560
5–9 years	12,000
10–14 years	9,000
15–59 years	57,000
60 years and older	7,000
Malnutrition rates (number and percent	
PEM	
Children younger than five	3,900 (30)
Adults stunted from childhood malnutrition	17,000 (30)
Iron	
Anemic children under age 15	18,000 (50)
Anemic adult men	7,250 (25)
Anemic pregnant women	2,520 (63)
Total population anemic	49,000
Iodine	
Population deficient	24,000 (24)[c]
Cretinism	50 (0.4)[d]
Vitamin A	
Deficient children under six	1,950 (15)
Severely deficient children under six	40 (.27)
Severely deficient children under six dying	20 (.16)
Partially blind children under six	81 (0.060)
Totally blind children under six	41 (0.028)
Annual deaths from malnutrition (number)	
PEM-related causes in children under five	160
Severe anemia in women at childbirth	10
Stillbirths related to iodine deficiency	10
Neonatal deaths related to iodine deficiency	10
Children under five with vitamin A deficiency	40
Degree of disability (percent)[e]	
Undernutrition	10
Iron deficiency	20
Iodine deficiency	5
Cretinism	50
Partial blindness	25
Total blindness	50

a. Includes coverage as well as efficacy.
b. Adults age 15–59.
c. Includes 25,000 women of reproductive age, of whom 4,000 are pregnant.
d. One child is born with cretinism each year.
e. Health and productivity disability.
Source: Based on authors' assumptions.

Table 19E-2. Nutrition Program Costs for Population of 10,000

Intervention	Target group	Annual per capita cost (U.S. dollars)	Annual program cost (U. S. dollars)
Food supplements	Pregnant women Children 0–3 years	46	620,540
Nutrition education	Pregnant women	2	26,980
Food subsidy	Bottom quintile	30	600,000
Integrated nutrition PHC	Pregnant women	25	337,250
School feeding	Children 5–9 years	12	144,000
Iron			
Supplement[a]	Pregnant women	2	8,000
Fortification	Entire population	0.20	20,000
Iodine			
Supplement, selective	Women	0.50	12,500
Supplement, total	Entire	0.50	23,250
Fortification	Entire population	0.10	10,000
Vitamin A			
Supplement	Children 0–5 years	0.50	6,500
Fortification	Entire population	0.20	20,000

Note: Based on assumptions in table 19A-8.
a. Assumes six prenatal visits plus 200 iron tablets.
Source: Ho 1985; Levin 1985; Kennedy and Alderman 1987.

Table 19E-3. Costs and Effectiveness of Iron Intervention

Parameter	Iron supplementation of pregnant women	Iron fortification
Target group	Pregnant women	All people
Number	4,000	100,000
Average rate (percent)[a]	63	50
Per capita cost (U.S. dollars)[b]	2	0.20
Program effectiveness (percent)	75	75
Deaths averted	10	10
Immediate productivity gains (percent)	20	20
Program duration (days)	200	Year round
Program costs (U.S. dollars)	8,000	20,000
Discounted wage gains (U.S. dollars)	221,280[c]	1,682,720[d]
DALY gained	624	4,520
Wage gains divided by program cost	27.7	84.1
Cost per DALY (U.S. dollars)	12.80	4.40
Cost per death averted (U.S. dollars)	800	2,000

Note: Based on assumptions in table 19E-1.
a. Rate of anemia for iron supplementation of pregnant women; rate of iron deficiency for iron fortification.
b. Per pregnancy for iron supplementation; per participant for iron fortification.
c. Calculated as the product of the number of anemic participants times disability times wage times effectiveness times employment, plus the product of number of deaths times wage times employment times productive life expectancy; ([0.63 x 3990] x 0.2 x 500 x 0.75 x 0.75) + (10 x 500 x 0.75 x 21.3) = 141,400 + 79,880 = 221,280.
d. Calculated as the product of the number of adult participants times the rate of anemia times disability times effectiveness times employment times wage, plus the product of the number of deaths times wage times employment times productive life expectancy; (56,990 x 0.5 x 0.2 x 0.75 x 500) + (10 x 500 x 0.75 x 21.3) = 1,602,840 + 79,880 = 1,682,720.
e. Calculated as the product of the number of deaths times life expectancy, plus the product of disability times number of malnourished participants times effectiveness; (10 x 24.7) + (0.2 x 0.63 x 3990 x 0.75) = 247 + 377 = 624.
f. Calculated as the product of number of adult participants times the rate of anemia times disability times effectiveness, plus the product of the number of deaths times life expectancy; (56,990 x 0.5 x 0.2 x 0.75) + (10 x 24.7) = 4270 + 250 = 4520.
Source: Based on authors' assumptions.

Table 19E-4. Costs and Effectiveness of Iodine Intervention

Parameter	Iodine supplement: targeted coverage	Iodine supplement: mass coverage	Iodization of salt or water
Target group	Reproductive-age women	Everyone under age sixty	Everyone
Number	25,000	93,000	100,000
Average rate of iodine deficiency (percent)	24	24	24
Per capita cost (U.S. dollars)[a]	0.50[b]	0.50	0.10
Program effectiveness (percent)	75	75	75
Deaths averted	10[c]	10	10
Productivity loss (percent)			
Normal population	5	5	5
Cretins	50	50	50
Program duration	Year round	Year round	Year round
Program costs (U.S. dollars)	12,500	46,500	100,000
Discounted wage gains (U.S. dollars)	172,000[d]	280,000[e]	280,000[e]
DALY gained	660[f]	1,270[g]	1,335[h]
Wage gains divided by program cost (U.S. dollars)	13.8	6.0	28
Cost per DALY (U.S. dollars)	18.90	37	7.50
Cost per death averted (U.S. dollars)	1,250	4,650	1,000

Note: Based on assumptions in table 19E-1.

a. Per participant per year. b. Prevents both neonatal death and cretinism. c. Neonatal.

d. Calculated as the product of the number of participants times the rate of deficiency times disability times wage times effectiveness times employment rate, plus number who died times productive life expectancy times employment times wage for ten cretins, plus the product of frequency times productive life expectancy times employment times wage for ten deaths; (25,000 x 0.24 x 0.05 x 500 x 0.75 x 0.75) + (10 x 0.5 x 15.5765 x 0.75 x 500) + (10 x 15.5765 x 0.75 x 500) = 84,380 + 29,210 + 58,410 = 172,000.

e. Calculated as in note d; (57,000 x 0.24 x 0.05 x 0.75 x 0.75 x 500) + (10 x 0.5 x 15.5765 x 0.75 x 500) + (10 x 15.5765 x 0.75 x 500) = 192,380 + 29,210 + 58,410 = 280,000.

f. Calculated as the product of the number of participants times the rate of deficiency times disability times effectiveness, plus the product of disability times life expectancy for ten cretins, plus the life expectancy for ten deaths; (25,000 x 0.24 x 0.05 x 0.75) + (10 x 0.5 x 29) + 10 x 29 = 225 + 145 + 290 = 660.

g. Calculated as in note f; (93,000 x 0.24 x 0.05 x 0.75) + (10 x 0.5 x 29) + 10 x 29 = 837 + 145 + 290 = 1270.

h. Calculalated as in note f; (99,980 x 0.24 x 0.05 x 0.75) + (10 x 0.5 x 29) + 10 x 29 = 900 + 145 + 290 = 1335.

Source: Table 19E-1 using methodology described in d (above).

Table 19E-5. Cost and Effectiveness of Vitamin A Intervention

Parameter	Vitamin A supplementation[a]	Vitamin A fortification
Target group	Children under five	Entire population
Number	13,000	100,000
Average rate of vitamin A deficiency (percent)[b]	15	15
Per capita cost (U.S. dollars)[c]	0.50	0.20
Program effectiveness (percent)	75	75
Deaths averted (number)	20	20
Blindness averted (number)		
Total	4	4
Partial	8	8
Productivity loss (percent)		
Totally blind	50	50
Partially blind	25	25
Program duration	Year round	Year round
Program costs (U.S. dollars)	6,500	20,000
Discounted wage gains (U.S. dollars)	140,188[d]	140,188[d]
DALY gained	696[e]	696[e]
Wage gain divided by program cost	21.6	7.0
Cost per DALY (U.S. dollars)	9.3	29
Cost per death averted (U.S. dollars)	325	1,000

Note: Based on assumptions in table 19E-1.

a. Semiannual mass dose. b. In children under five. c. Per participant.

d. Does not include losses due to excess child morbidity. Calculated as the product of the number of deaths averted times the productive life expectancy times employment times wage, plus the product of the number of total blindness averted times productive life expectancy times disability times employment times wage, plus the product of the number of partial blindness averted times productive life expectancy times disability times employment times wage; (20 x 15.5765 x 0.75 x 500) + (4 x 15.5765 x 0.5 x 0.75 x 500) + (8 x 15.5765 x 0.25 x 0.75 x 500) = 116,824 + 11,682 + 11,682 = 140,188.

e. Calculated as deaths averted times discounted remaining life expectancy plus total blindness times disability times discounted remaining life expectancy plus partial blindness times disability times discounted remaining life expectancy; (20 x 29) + (4 x 0.5 x 29) + (8 x 0.25 x 29) = 696.

Source: Based on authors' assumptions.

Notes

1. In developing countries, 50 percent of children from birth to fourteen years, 25 percent of adult men and women, and 63 percent of pregnant women are iron deficient; 24 percent of the total population are iodine deficient, and 0.4 percent is cretinous because of iodine deficiency; 15 percent of children under five are deficient, 2.5 percent are severely deficient in vitamin A, 1.4 percent die of severe deficiency, 0.28 percent become totally blind, and 0.66 percent become partially blind.

Thirteen percent of the population of developing countries are under five years of age; 21 percent are five through fourteen; 57 percent are economically active adults, 44 percent of whom are women of reproductive age of whom 16 percent are pregnant; 7 percent are sixty years of age or over. Adults were assumed to be economically active between fifteen and fifty-nine years of age.

Excess mortality due to severe vitamin A deficiency was assumed to be 1 percent of the target age group, or about 41 percent of the severely deficient children; excess mortality due to iron deficiency was assumed to be 1 out of 400 pregnant women; excess mortality due to iodine deficiency was assumed to be 1 neonatal death out of 96 pregnancies of iodine-deficient women.

Partial blindness: 25 percent disability; total blindness: 50 percent disability; iron deficiency: 20 percent disability; iodine deficiency: 5 percent disability; cretinism: 50 percent disability.

Iron supplementation: $2.00 per pregnancy; iron fortification: $0.20 per capita per year; iodine supplementation: $0.50 per recipient per year; iodine fortification: $0.10 per capita per year; vitamin A supplementation: $0.50 per recipient per year; vitamin A fortification: $0.20 per capita per year.

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